A CLINICAL STUDY ON

MADHUMEGAM (DIABETES MELLITUS)

WITH THE EVALUATION OF SIDDHA DRUG

PUNGAMPOO CHOORANAM

The dissertation submitted by

Dr. J. NISHA

Reg. No. 321411108

Under the Guidance of

Prof. Dr. N. Anbu, M.D. (S)

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements

For the award of the degree of

SIDDHA MARUTHUVA PERARIGNAR

DOCTOR OF MEDICINE (SIDDHA)

BRANCH I - MARUTHUVAM

POST GRADUATE DEPARTMENT OF MARUTHUVAM

THE GOVERNMENT SIDDHA MEDICAL COLLEGE

CHENNAI – 106

OCTOBER - 2017

CERTIFICATE

This is to certify that the dissertation entitled **" A CLINICAL STUDY ON MADHUMEGAM "** is a bonafide work done by **Dr.J.NISHA**, Government Siddha Medical College, Chennai – 600 106, in partial fulfilment of the university rules and regulations for award of '**SIDDHA MARUTHUVA PERARIGNAR'** under my guidance and supervision during the academic year 2014 – 2017.

Name & Signature of the Guide

Name & Signature of the HOD

Name & Signature of the Principal

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CONTENTS

CONTENTS

INTRODUCTION

INTRODUCTION

Siddha System of Medicine is one of the ancient systems practiced in South India especially in Tamilnadu, and this system is contemporaneous with those of the Egyptian, Mesopotamian, Chinese and Grecian medicines. The aim of Siddha medicine is to make the body perfect, imperishable and to promote longevity.

The unique nature of this system has its continuous service to humanity for more than 5000 years in combating diseases and in maintaining its physical, mental and moral health. [1]

The word ‘Siddha’ has its origin in the Tamil word Siddhi which means "an object to be attained" or "Perfection" or "Heavenly Bliss". [2]

Traditional Siddha medicine upholds balancing, up righting and eliminating the pathogens as the main principles of treating diseases and maintaining health. Siddha Medicine gives equal importance in prevention and curing. [3]

Siddha system is mainly based on ‘Andapinda Thathuvam’ that means the relationship between the universe and human body. These two are interlinked through the five basic principles which are known as ‘Panchaboothas’. The structural aspect of the human body is said to be ‘Udal Thathus' (i.e. the physical component of the human body) and the functional units of the human body is said to be ‘Uyir Thathus’ (the physiological units i.e., Vatham, Pitham, and Kapham). Functional co-operation of these two are essential for the maintenance of health. [4]

Yugi Muni describes 20 types of Meganoigal, Madhumegam is one among them.

The word Madhumegam is very similar and closely resembles with the chronic metabolic disorder called Diabetes Mellitus-Type II in Modern medicine.

Susruta, known as the Father of Indian Medicine, described many of the symptoms of Diabetes in his ‘Samhita’ and called the affliction "Madhumegam" based on the sweetness of the urine passed by those afflicted by this ailment.[5]

Ebers papyrus may be the first recorded evidence about Diabetes, it seems to be more of historical importance as it was only discovered in the last century. [6]

Madhumegam is a group of metabolic disorder in which a person has high blood sugar, either because the pancreas does not produce enough Insulin, or because cells do not respond to the Insulin that is produced. This high blood sugar produces the classical symptoms of Polyuria (frequent urination), Polydipsia (increased thirst) and Polyphagia (increased hunger). [7]

WHO recently compiled data show that approximately 150 million people have Diabetes Mellitus worldwide and that this number may well double by the year 2025. Much of this increase will occur in developing countries and will be due to Population growth, Ageing, Unhealthy diets, Obesity and Sedentary lifestyles. By 2020, India is expected to be the Diabetes capital of the world. [8]

Most studies report worse quality of life for people with Diabetes compared to the general population, especially regarding physical functioning and well-being.

Unfavourable modification of lifestyle and dietary habits that are associated with urbanisation are believed to be the most important factors for the development of diabetes. The prevalence of diabetes is approximately twice in urban areas than in rural population. [9]

Siddha medicine is an answer to all obstinate diseases and chronic sufferers, especially for treatment of Diabetes Mellitus-Type II. Instead depending totally upon modern medicines, one can try natural treatment like Siddha medicine for management of Diabetes Mellitus. Such natural medicines also strengthen the Pancreas and regulate smooth flow of Insulin.

Siddha is an effective natural treatment modality for Diabetes management. The Herbal remedies of Siddha helps to reduce blood sugar level down and enhance glucose tolerance. There are many Siddha medicines that can be used for the treatment of Diabetes Mellitus Type II and “Pungampoo Chooranam” is one of the Siddha medicines which have the proven results to reduce the blood Sugar level.

On the whole as a compound drug ‘Pungampoo Chooranam’ will be an effective as well as Biosafe drug in the treatment of ‘Madhumegam’.

AIM
AND
OBJECTIVES

AIM:

The Aim of this study is to evaluate the Clinical Efficacy and Safety of Siddha Medicine “Pungampoo chooranam’’ in the management of Madhumegam.

OBJECTIVES:

1. To review the Siddha literary evidences dealing with Aetiology, Classifications, Signs & Symptoms, Diagnosis, Diet and Prognosis of Madhumegam in Siddha system of Medicine.
2. To study Madhumegam in various literatures in comparison with Diabetes Mellitus -Type II.
3. To understand the incidence of the disease with reference to Age, Sex, Thinaigal, Paruvakalam, Socio Economic conditions, Diet and Family history.
4. To explore the unique diagnostic methods mentioned by Siddhars such as Envagaithervu, Mukkuttram, Udal thathukkal with specific reference by Naadi, Neerkuri and Neikuri.
5. To implement Siddha and utilize Modern parameters to diagnose and to confirm the severity & progress of the disease.
6. To evaluate the Bio-chemical analysis of the trial drug.
7. To assess the Acute and Sub- Acute toxicity of the trial drug.
8. To study the Anti Diabetic activity of the trial drug.
9. To evaluate the Clinical study of the trial drug.
10. To analyze the Biostatic analysis of the trial drug.

REVIEW OF LITERATURE

SIDDHA ASPECTS

REVIEW OF LITERATURE

SIDDHA ASPECTS

“தக்க தாரணி மானிடத்தோர்கள் கேள்
பக்கமாசலம் பத்திருவகையுமே
நக்க நாயகன் நாயகிக்கே சொல்
மிக்க நந்தி விளம்பி விதித்ததே”

– தேரையர் வாகடம் [10]

According to Therayar Vagadam, “The universe consist of two essential entities that is, matter and energy which Siddhar’s referred to as Shiva and Sakthi”. Shiva explained Megarogam to Sakthi. Here Nandhi explains its symptoms to the world for the benefit of the human kind. This clearly indicates that the existence of this disease is as old as human race.

“ஆமப்பா மனிதர் செய்த கன்மத்தாலே
அரகரா மேகமென்ற ராசாவாலே”

– அகத்தியர் [11]

According to Siddhars, the imbalance of tridosha causes totally 4448 diseases to human beings. Among them, Megarogam is considered to be the emperor of diseases.

“மிகினும் குறையினும் நோய்செய்யும் நூலோர்
வளி முதலா எண்ணிய மூன்று”

– திருக்குறள் [12]

All the diseases are due to alteration of three vital humours and seven physical constituents.

The factors, which affect this equilibrium of vital humours are,

- Unavu Marupadugal (Altered diet habit)
- Kalamarupadugal (Seasonal variations)
- Thega vanmai (Depending upon immunological status)

Madhumegam has its description in various literatures like Yugi Vaithya Chindamani, Agasthiyar Gunavagadam.

Earlier, diseases were classified only according to Mukkutram. Yugi Munivar classified the diseases according to cause, signs and symptoms, and also explained about the prognosis, treatment and diet, which is now followed by the modern world.

VERUPEYARGAL (SYNONYMS)

Neerizhivu, Ennipuneer, Vegumoothiram, Thithippuneer, Miguneer. [13]

Neerizhivu – Excess of urination.

Ennipu Neer – The urine is sweet in taste.

IYAL (DEFINITION)

Madhumegam is a clinical condition characterized by frequent passage of urine more than the normal resulting in deterioration and diminution of the seven thathus.

‘‘அண்மையாயடிக் கடிக்கு நீரிறங்கு
மடிக்கடிக்கு அரைநாழி தனிலே காணும்
வெண்மையான தடிய தனிற்றான் பிடிக்கும்
மிக்கான சடம்வெளுத்து மேனிகன்றும் ’’

– யூகி வைத்திய சிந்தாமணி [14]

These lines quote frequent micturition, more than the normal with large quantity resulting in detoriation of gradual dimnision of sevan udal thathukkal.

’’நீரினைப் பெருக்கலென்று நீரிழி விலக்கணங்கேள்
நீலவாரிதி போற் குக்கி நீட்டிக்கு முரை தள்ளாகும்
நீவி கூடாது கை, கால் நீலமா வினை நேராகும்
நீள் சொனாவுரனின் மூச்சு நீசமா முயங்கக்காட்டும்’’

– தேரையர் மகா காரிசல் [15]

Abdomen distends like sea, slurring of speech, peripheral neuritis, lassitude, dyspnoea are the symptoms of Madhumegam.

As per Athma Rakshamirrtham, body becomes weak, weight loss, dryness of skin and tongue, excessive thirst, tiredness, excess sleep indicate the presence of Megaroham.

NOI VARUM VAZHI-AETIOLOGY

"மேகமெனு நீரழிவு வரும் விதத்தை
விளம்புகிறேன் முன்செய்த கர்மந்தன்னால்
தாகமுடன் மதுபதார்த்தங்கள் நன்றாய்த்
தான்புசித்த லாலுஞ்சிற்றினத்தின் மங்கை
போகமதி கரித்தலா லுட்டினந்தான்
போதவே மிஞ்சுதலால் தயிர்மோர் நெய்பால்
ஏகமாய்ப் புசித்தலாற் கொழுத்த வூனை
யென்று முண்ண லுவந்நீரைக் குடித்தலாலே
ஆசையுடன் சிறுவழுதலங்காய் தன்னை
யதிகமா யுண்பதால் காலந்தப்பில்
போசனங்கள் செய்தலால் நடையலைச்சல்
போதவே யிருத்தலிரா கண்விழித்தல்
தேசமெங்கு திரிதலா மிவைகளாலே
சிரந்தனிற்சூ டதிகங் கொண்டுடனே ரத்தம்
சோஷிதே யதிகமாய் மேகந்தோன்றித்
தொல்லை செய்யும் நீரழிவும் இருபதாமே."

– சரபேந்திர மேகநிவாரணபோதினி என்னும் நீரிழிவுநோய் மருத்துவம். [16]

(i) Diet Habits

"கோதையர் கலவி போதை
கொழுத்தமீ னிறைச்சி போதை
பாதுவாய் நெய்யுண் பாலும்
பரிவுட ண்பீ ராகில்
சோதபாண் டுருவ மிக்க
சுக்கில பிரமே கந்தான்
ஒதுநீ ரிழிவு சேர
உண்டென வறிந்து கொள்ளே"

– அகத்தியர் 1200. [17]

"உற்பவிக்கும் பால்நெய்யா லிறைச்சி கொள்ளல்
உரிசையாய் மீன்றன்னா ல்வருவி ருத்த
மற்பவிக்கும் பதார்த்தத்தால் மதுர வஸ்தால்
மந்தங்கள் தனைபொசித்தல் வேகாப் பண்டம்
குற்பவிக்குங் குளுந்தவன்ன மங்கை கோஷ்டி
குறித்த நித்திரைதவிர் தலக்கினி மந்தம்
துற்பவிக்குஞ் சரீரந்தான் மிகப்பருத்தல்
சஞ்சலந்தான் பயப்படுதல் தரிக்கும் நோயே"

– யூகி வைத்திய சிந்தாமணி [18]

Excessive intake of food, rich in carbohydrate and fat, red meat, sweet food, rawfood and sleeplessness induces mathumegam, which was quoted by Agathiyar and Yugi Munivar.

(ii) Sexual Indulgence:

"கன்னி மயக்கத்தால் கண்டிடு மேகமே"

– திருமூலர் [19]

"கிரந்திப் புண்ணிரண மேகக்
கீசக யெனன்னுந் துன் மார்க்கன்
அருந்ததி யென்னும் பாஞ்சாலி யன்னையைக்
கண்ணுற்றானே"

– தேரையர் மருத்துவ பாரதம் [20]

According to Thirumoolar and Therayar, excessive indulgence in sex causes Megaroham.

(iii) Obesity:

"தற்பிவிக்குஞ் சரீரந்தான் மிகப்ப ருக்கல்
சல்சலந்தான் பயன்படுதல் தரிக்கும் நோயே"

– யூகி வைத்திய சிந்தாமணி [21]

Obesity is one of the main cause of Madhumegam.

(iv) Psychosomatic Cause:

"இயம்பவே ஆறுகுளம் பின்னஞ் செய்தல்
ஏற்றமாய் மாற்றான்பெண் சங்கம் செய்தல்
பயம்பவே பாலகர்களுக் கொளித்து தின்னல்
பழமை சலம்போறவ ழிதனை தடுத்தல்
அயம்பவே ஆலயத்திற் சலம்விட்டோர்க்கும்
ஆதியாம் வேதத்தைத் தூஷித் தோர்க்கும்
துயம்பவே சூரியனை வணங்கா தார்க்கும்
சுருக்காக மேகம்வந்ததுற் பவிக்குந் தானே"

– யூகி வைத்திய சிந்தாமணி [22]

According to Yugi Vaidya Chinthamani, Megaroham may occur due to not giving proper respect to Guru, Father, Mother, Vedas and God suriyan.

(v) Hereditary:

"முறைகேட்கின் ஒன்பது முயற்சியால் வந்தது
துறை கேட்கிற கருப்பத்திற் றுவங்கிய மேகங்கள்
நிறை பூத்த கொங்கையாள் நாயகன் மோகத்தால்
மறை போற்றுங் கருப்பத்தில் வளந்ந்தது."

– அகத்தியர் வைத்திய காவியம் [23]

Thirumoolar have noted in his literatures that Hereditary is one among the causes of the disease Madhumegam. At present researches have also found that genetic factors play an important role in Madhumegam.

(vi) Excess Stimulation of Moolatharam:

"சரியானமேகத்தா லபான வாயு
தான் புகைக்கு மேலேறிக் கபாலச் சூடாம்
பெரிதான மேகத்தா லத்தி வெந்து
போமப்பா தசைவெந்து ரத்தம் வற்றிப்
பரிவாகித் தச வாய்வால் மந்தம் கொண்டு
பெருந்தீனி மலபந்தம் உதான வாயு
விரிவாகித் தேகமெல்லாம் விட ரூராலே
மெய்யழிந்த தேகமென்ற திருபதாச்சே"

– நோய் நாடல் [24]

Among the six Atharams, the Moolatharam is situated in between rectum and genitals, just end of sacral plexus.

In the Madhumegam disease, impaired Abana vayu (excretory junction) inactivate the moola agini during that time excess intake of food causes inactivation of dhasavayu which create excessive appetite (Polyphagia) and constipation. Udanan is also affected. These changes in turn cause the derangement of seven udal thathukal.

(vii) Deeds:

"தானே பூருவ விதியினால் சாரும் பிணிக ளெல்லாம்
மானேர் விழியாள் வேட்கையினால் வருந்தும் பின்னும் பசியால்
தானே பொறுத்து உண்கையினால் தாகந்தன்னால் மிகச்சோர்ந்து
தானே கமலம் புண்ணாகி செய்யும் பிரமேகச் செயல்தானே"

– தேரையர் மருத்துவ பாரதம் [25]

From the above poem, the diseases also occur as a result of bad deeds committed in previous or this birth.

MURKURIGUNAM (PREMONITORY SYMPTOMS):

Premonitory symptoms of Madhumegam are polyuria, polyphagia, poly dipsia. Madhumegam exhibits the following premonitory symptoms from its initial stage of development itself. The patient experiences voracious hunger, thirst, perspiration, exhaustion and giddiness. The excessive intake of water to quench thirst is excreted as excessive quantity of urine (poly uria). In spite of abnormal consumption of food, stamina continues to decrease.

- NoiNadal [26]

PODHUKURIGUNANGAL (GENERAL SIGNS & SYMPTOMS)

‘‘கூறான மேகமது இருபதுக்கும்
குணந்தன்னைச் சிவன்சொல்ல தேவி கேட்க தாறான
தாகமொடு சோகமேகந்
தரியாமல் நீரிழித விருமல் மூச்சு
ஆறான அருசிசத்தி சித்தப் ரம்மை
அடிகடிக்கடிக்குத் தண்ணீந்தான்அங்கே கேட்கல்
ஈறான இடுப்புக்குள் கடுப்பு காணல்
எலும்புழற்ற லழற்றல டெரிவு டாமே
எரிவோடு சரீரமெல்லா மறைபட்டாற்போல்
எலும்பு நோதல் நித்திரையில் லாமை
வரிவோடு மாய்விமெத் தவும்பறித்தல்
மனதுசஞ் சலப்படுதல் காற்று வேண்டல்
மெரிவோடு மேல் மூச்சு மிகவுண்டாதல்
விக்கலொடு மயக்கந்தான் மெத்தக் காணல்
தெரிவோடு தேகமெங்கும் வெளுருண்டாதல்
தேகமெத்த வாலோபப் படுதல் காணே’’

– யூகி வைத்திய சிந்தாமணி [27]

Common Symptoms:

Thirst	Polyuria
Polydipsia	Cough
Anorexia	Dyspnoea
Delirium	Pain in the hip and burning sensation
Sleeplessness	loss of weight
Hiccough	Flatulence
Anaemia	Giddiness

NOI VAGAIKAL - (CLASSIFICATION)

Megarogam is classified into twenty varieties to quote from Agasthiar.

‘‘உட்டிண ரோகத்தாலும் உறும்பெரும்
பசியினாலுங் கட்டவிழ் கோதை மாதர்
கலவிமட்டிலா மையலாலு முட்டறா நாலுமாறு மும்
மூன்று மொன்று மொன்று திட்டமாய்
வருவதென்று திருமுனி யருளிச் செய்தார்’’

–அகத்தியர் [28]

Yugi Munivar classifies the same as

‘‘வசனித்த மேகமது யிரண்டு பத்து
வாதத்திற் பிறந்தசலம் நாலேயாகும்
பிசனித்த பித்தத்திலு ற்றபவித்த
பேராசை லந்தானு மாறு மாகும்
தேசனிந்த சேட்டுமத்திலுற்ப வித்த
சீரான சலந்தானும் பத்தேயாகும்’’

– யூகி வைத்திய சிந்தாமணி [29]

According to Theraiyar

“கழியும் வாதம் நான்காலும் காயும் பித்த மாறாலும்
சுழியும் சேத்துமண் பத்தாலும் சொல்லும் நாலஞ்சாய் தோன்றும்

– தேரையர் வாகடம் [30]

NOI VAGAIGAL:

BOOKS	NOI ENN	VALI	AZHAL	IYAM
யூகி வைத்திய சிந்தாமணி	20	4	6	10
அகத்தியர் 1200	20	4	6	10
தேரையர் வாகடம்	20	4	6	10
தன்வந்திரி வைத்தியன்	20	4	6	10
சரபேந்திர நீரிழிவு ரோக சிகிச்சை	20	4	6	10

The above books describe twenty different kinds of megam (urinary disorders) on the basis of colour, consistency, taste, smell, weight etc.

Out of these twenty different kinds four varieties are caused by Vali, six varieties are caused by Azhal, ten varieties are caused by Iyam.

Madhumegam comes under the classification of Azhal.

CLASSIFICATION OF MEGAM:

According to Yugi Vaidhya Chinthamani,

- **Vaadha Neer Vagaigal:**

''தரித்திட்ட வாதத்தின் சலந்தா னாலு
தனியான நாலுக்கும் பேரே தென்னில்
அரித்திட்ட ஆச்சியகெந்தி மேகத்தோடு
அதன்பிறகு சுற்றமா மேகமென்று
பிரித்திட்ட பிரமிய மேகமென்று
பேரான மாங்கரவி மேகமென்று''

– யூகி வைத்திய சிந்தாமணி [31]

Vali – 4

1. Neimananeer
2. Pasumana neer
3. Seezhmana neer
4. Sathaimana neer

- **Pitha Neer Vagaigal:**

"முறையான பித்த சல மாறுமாகும்
முதிர்ந்த அப்பிய மென்றும் பிரமிய மென்றும்
துறையான சாம்பீர்ணமதும்ப மென்றும்
சாத்திகமே யாறுவிதந் தன்னோ டாறு"

– யூகி வைத்திய சிந்தாமணி [32]

Azhal – 6

1. Yanai kozhupu mana neer
2. Katrazhai mana neer
3. Chunna mana neer
4. Innipu megam
5. Palingu neer
6. Muyal kurithi neer

- **Iya Neer Vagaigal**

"ஆறான சிலேட்பசலம் பத்து தன்னை
அரன் சொல்ல ஆத்தாள் தான் கேட்கும் போது
வாறான வசாமேகம் உத்சமேகம்
மச்சியாமே கத்தோப கீத மேகம் தூறான சுராரி சுக்ல முத்த
மேகம் சுற்றமாம்பி னானியொட வலண மேகண்
கேறான தெயுத்தயமா மேக மென்று
செப்பினார் சிலேட் பத்தின் செலுத்துத் தானே"

– யூகி வைத்திய சிந்தாமணி [33]

Iyam – 10

1. Iaya Neer
2. Thuimai Neer
3. Moolai neer
4. Ilaneer
5. Kal neer
6. Thavala Neer
7. Kazhu neer

8. Then neer
9. Uppu neer
10. Kavichi Neer

Yugi described four types under the Vatha premeham, six types under the pitha prameham and ten types under Kaba prameham**.**

NOIKURI KUNANGAL – (CLINICAL FEATURES)

Polyuria, Polyphagia, polydipsia, perspiration, exhaustion, insomnia, giddiness and loss of weight are seen even at normal consumption of food.

COMMON SIGN AND SYMPTOMS OF PITHA PRAMEHAM:

‘‘அறியவே பித்தசல மாறுக்குந்தான்
அங்கமதிற் செய்கின்ற குணத்தைக் கேளாய்
தறியவே சரீரம் வற்றி யெரிவுங்டாகும்
சடத்திலுந் நீரிலுந்தான் கவிச்சுண்டாகும்.
தெறியவே சீப்போலுங் கற்றாழை போலும்
சேல் போலுந் தேன் போலும் நாற்ற முண்டாம்
வெறியவே பீசத்திற் கோசத்திற் குதத்தில்
மிகுமீரல் நாபியிலும் வேக்கா டாமே
வேக்காடாய் விரண முண்டாய் வாய்தான் நாறும்
விக்கலொடு அருசியாயச் சுரமுண்டாகும்
தீக்காடாய்த் தேகந்தான் கிடைகொட்டாது
தியக்கமொடு மூர்ச்சையுண்டா மயக்க மாகும்
சாக்காடாய் நாவறந் தண்ணீந் தாகம்
சக்தியொடு சரீரமெல்லாந் தளர்ச்சி யாகும்
தாக்கடா மலசஞ்சலந்தான் மிகவுண்டாகும்
சமகுணந்தான் பித்த சல மாறு மாச்சே’’

– யூகி வைத்திய சிந்தாமணி [34]

As per the above poem, polyuria, polyphagia, Polydipsia, fever, angular stomatitis, burning sensation all over the body, loss of weight, are common signs and symptoms of pitha prameham.

MUKKUTRA IYAL:

"வாதமாய் படைத்தது பித்த வன்னியாய் காத்து

சேத்தும சீதமாய் துடைத்து"

– தேரையர் மருத்துவ பாரதம் [35]

- **Vali:**

Sites of Vadha:

Below Naval, Urinary bladder, intestines, Pelvis, umbilical cord, thigh, bone, skin, nerve endings, joints, Musculature, hair root.

Properties:

Dryness, Lightness, Clearness, Coolness, Mobile, Formless.

Function:

- Praanan (Uyirkaal) :

  This controls knowledge, mind and five sense organs, which are useful for breathing and digestion.

- Abaanan (Keezh nokku kaal) :

  This is responsible for all down ward movements such as passing urine, stools, semen, menstrual flow etc

- Samaanan (Nadukkaal) :

  This aids in proper digestion.

- Viyaanan (paravukaal):

  This is responsible for all movements of all parts of the body.

- Uthaanan (Mel Nokkukaal) :

  Responsible for all upward visceral movements, such as vomiting, eructation and nausea.

- Naagan :

  Responsible for opening and closing the eyes.

- Koorman :

  Responsible for vision and yawning.

- Kirukaran :

  Responsible for salivation, nasal secretion and appetite.

- Devathatthan :

  Responsible for Laziness, sleeping and anger.

- Thananjeyan :

  Produces bloating of the body after death. It escapes on the third day after death bursting out of the cranium.

In Madhumegam

- Piranan : Normal
- Abanan : Constipation, Noctural polyuria, frequency of micturation.
- Viyanan : Symmetrical sensory disturbances, peripheral neuritis, pain all over the body, Burning sensation in the sole of foot and palm, Skin infection and carbuncle.
- Udanan : Normal
- Samanan : Poly Phagia
- Nagan : Normal
- Koorman : Diabeic Retinopathy, Cataract
- Kirukaran : Poly Phagia
- Devathathan: Normal
- Thananjeyan: -

- **Azhal:**

Sites of Pitha:

Between the heart and the naval, sweat, lymph, blood, stomach, urinary bladder, heart, saliva, eyes and skin.

Properties:

Dry, cold, hot, light, subtle, keen, soft, liquid, bitter.

Function

1.	Anal Pittham	: It promotes appetite and helps in digestion.
2.	Ranjagam	: It gives colour to the blood.
3.	Praasagam	: It gives complexion to the skin.
4.	Aalosagam	: It brightens the eyes.
5.	Saathagam	: It controls the whole body. It has the property to fulfil all the activities which the mind desires.

In Madhu megam

Anala Pitham	-	Excess hunger
Ranjaga pitham	-	Pallor sometimes
Alosagapitham	-	Dimness of vision
Saathaga pitham	-	Lassitude
Prasaga pitham	-	Dry skin

- **Iyam:**

Sites of Kapha:

Above the heart, stomach, fat, sperm, tongue, uvula, bone marrow, blood, nose, nerves, bones, large intestine, eyes, joints.

Properties:

Heavy, cold, mild, watery, sweet and stable.

Function:

) Avalambagam	:	Lies in the lungs, controls the heart and other kabhams.
) Kilethagam	:	Lies in the stomach, makes the food moist, soft and helps in digestion.
) Pothagam	:	Responsible for identifying taste.
) Tharpagam	:	Present in the head and responsible for the coolness of eyes.
) Santhigam	:	Responsible for lubrication and free movements of joints.

In Madhumegam

Avalambagam	-	Normal
Tharpagam	-	Burning sensation in the eyes
Santhigam	-	Joint pain
Kilethagam	-	Excessive appetite
Pothagam	-	Normal

SEVEN UDAL THATHUKKAL (PHYSICAL CONSTITUENTS)

Annamaya kosa is constituted by seven Thathus. They are the basic tissues of our body.

Normal functions:

- **Saram:**

  It is responsible for the growth and development. It keeps the individual in good spirit and it nourishes the blood.

- **Senneer:**

  Blood imparts colour to the body and nourishes the muscle responsible for the ability, intellect of the individual.

- **Oon:**

  It gives shape to the body according to the requirements for the physical activity, nourishes fat.

- **Kozhuppu:**

  It helps in lubricating the different organs and maintains only matter of the body.

- **Enbu:**

  Supports the system and responsible for posture and movements of the body.

- **Moolai:**

  It fills the bony cavity, nourishes semen, imparts strength endurance and shining appearance.

- **Sukkilam / Suronitham:**

  It is responsible for reproduction. In healthy people, they function in a harmony, while in diseased people, they are deranged.

In Madhumegam:

Saaram	:	Tiredness, General weakness
Senneer	:	Pallor
Oon	:	Emaciation
Kozhuppu	:	Dry skin
Enbu	:	Later stage due to infection it affects the bone and sometimes leads to amputation.
Moolai	:	Affected in Chronic stage.
Sukkilam / Suronitham	:	Impotence, Sexual urge is reduced.

So, in Madhumegam, Seven Udal Thathukkal are deranged.

MUKKUTRA VERUPADUGAL (PATHOGENISIS):

The disease Megaroham, due to external (or) internal causes affects balance in the ratio of vali, Azhal, Iyam. This imbalance affects the Keelnokkukal, which inturn affect the seven udal thathukkal. Saram gets affected and there is loss of appetite. Seeneer also get affected with the net result even if the patient eats more nourished food (polyphagia) there won't be any improvement in health.

An imbalance in pitham does imply an imbalance in other two kutrams too and causes derangement of dasa vayu and seven udal thathukkal which cause the disease and other complications.

PINIARI MURAIMAI- (DIAGNOSIS):

Diagnostic methods in Siddha system are very unique and solely based on clinical acumen of the physician.

- **Poriyal Arithal** (or) understanding by the five organs of perception (Mei, Vai, Kann, Mooku, Sevi).
- **Pulanal Arithal** (or) understanding by the sense objects (Uraithal, Suvaithal, Parthal, Mugarthal and Kettal)**.**
- **Vinadal** (or) Interrogation.

Tools used by Siddha Physicians:

- Kanndal (Perception)
- Karuthal (Inference)
- Urai (The instruction of the inspired)

The application of these three is very extensive in diagnosis and treatment.

ENN VAGAI THERVU (EIGHT TOOLS OF DIAGNOSIS)

- **Naa:**

Colour of the tongue, size, shape, anomalies, surface, mobility and local lesion should be noted. Coating deposition of the tongue, increased salivation and dryness of the tongue.

In Madhumegam, the tongue remains dry and at times black.

- **Niram:**

Colour of the skin all over the body, a local region of affection, conjunctiva, tongue, nail bud and hair etc.

Vatha Udal	-	Black and Whitish colour
Pitha Udal	-	Yellow or Reddish colour
Kabha Udal	-	White or Golden colour
Thontha Udal	-	Mix of two udal colours.

In Madhumegam, the colour of skin is different from original complexion, discoloured.

- **Mozhi:**

Observation of speech and voice.

In uncontrolled Madhumegam which leads to cerebrovascular disorder,

speech disorder sets in**.**

- **Vizhi:**

Colour, character, vision should be observed.

In uncontrolled Madhumegam cataract set in last. In longstanding cases, the Madhumegam affects retina and causes diabetic retinopathy which is the major cause of blindness.

- **Sparisam:**

  Colour of the skin (Vali, Azhal, Iyya udal), Eruption, Hemorrhages, Ulcers, Boils, trophic changes in the skin can be identified.

  Any changes in the internal organs can be noted by palpation (or) percussion.

  In Madhumegam, increased tendency for fungal infection like moniliasis and vulvities.

  In Madhumegam the skin is dry and pale.

- **Malam:**

  Quantity, colour, smell, froth should be observed.

  In Madhumegam, constipation sometimes yellowish loose stool are passed.

- **Muthiram:**

  Quantity colour froth smell and specific gravity of urine should be noted.

- **Urine:**

  Colour: In Madhumegam it is clear and white.

  Specific Gravity: In Madhumegam, urine is thick in consistency like honey.

  Smell: Honey like smell

  Froths: In Madhumegam the urine is frothy at the time of urination.

  Deposits: In Madhumegam few epithelial cells are present in urine.

  Normal quantity of adult urine is 750 – 2500 ml in 24 hours.

  Disturbing Polyuria at night (nocturia) and Glucosuria (the presence of sugar in urine) are present.

NEER NIRAKKURI:

‘‘அருந்து மாறிரதமும் அவிரோதமதாய்
அஃகல் அலதல் அகாலவூன் தவிர்ந்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியே காது பெய்
தொருமுகூர்த்தக் கலைக்குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே.’’

– நோய் நாடல் [36]

Collection of Sample Urine:

The patient must take well cooked food in the previous day. The intake must be proportionate to the degree of his appetite. Food intake should be taken, at appropriate time. The patient must have sound sleep on the previous night. The urine is collected on the dawn of the next day in a glass container and closed immediately to prevent contamination. This specimen must be examined with in one and half hours. This procedure should be followed strictly to get accurate observation of Neerkuri and Neikuri**.**

NEIKKURI:

‘‘நிறக்குறிக் குரைத்த நிருமாண நீரிற்
சிறக்க வெங்ணெய்யோர் சிறுதுளி நடு விடுத்
தென்னுறத் திறந்தவெளி யேகா தமைத்ததி
னின்றதிவலை போம் நெறிவிழியறிவும்
சென்றது புகலுந் செய்தியை யுணரே.’’

– நோய் நாடல் [37]

The diagnosis and prognosis of deranged Mukkutrams are studied on the basis of the behaviour of a drop of gingelly oil gently droped on the surface of the urine kept in a wide vessel in the sunlight.

‘‘முத்தொத்து நிற்கின் மொழவதென் கபமே.

– நோய் நாடல் [38]

In Madhumegam, the oil dropped in urine is like a pearl and if the oil spreads slowly, the prognosis of the disease is slow and good.

NAADI:

Pulse is the confirmatory diagnosis.

In Madhumegam,

"இருமியே பித்தமும் வாதமும் கூடிடில்
மருவுசல மேகம் வாருதி போலாகும் உருவம்
வேறாரு முண்டவுடன் காந்திடும் உருகவே
வூனோடு உறிஞ்சி இனிக்குமே"

– திருமூலர் [39]

Coupling of Azhal and vadha naadi causes excessive urine as vast as sea, loss of weight and polyphagia .

When kabha merges with vadha ,glucosuria,emaciation ,anaemia develops.

"பார்த்திடு மூன்றும் பதிந்து மெலிந்து நிற்கில்
தேர்ந்திடு மே வந் தோன்றியே பொருந்திமெய்யில்"

– திருமூலர் நாடி [40]

When all the three nadi's, runs in low volume,diabetes develops.

MADHUMEGA GUNAM:

"இனிக்கின்ற வாதத் திடைசேரில் ஐயந்தான்
பனிக்கின்ற கள்ளுப் பதனிபோல் நீரோடும்
கனிக்கின்ற மேனி கரைந்து வெளுப்பேறும்
தனிக்கும் மதுமேகந் தப்பாது ஐயனே"

– திருமூலர் [41]

The coupling of the Vali and Iya naadi, causes increase in urination, and diabetes develops.

AVATHAIGAL :

If the disease is not controlled or left untreated, the below complications follow gradually.

அவத்தை – 1

"காணவே முதவலத்தைச் சரீரந் தானும்
கனமாகப் பருத்திறுகி நீர்த்து வாரம்
வேணவே வெண்டாக்கிய கலம் பண்ணும்"

– சித்த மருத்துவம்

Obesity sets in.

There is obstruction in urinary flow.

Urinary passage expands due to inflammation.

அவத்தை – 2

" மிக்க இரண்டாமவத்தை விளம்பக்கேளாய்
மூணவே மூத்திரப் பீடையுமாச் சுக்கில
முகமழுகித் தேஜசுதான் மிகவே குன்றும்."

– சித்த மருத்துவம்

Bad odour while Micturation.

Loss of complexion in face and body.

அவத்தை – 3

"நாணவே மூன்றாகு மவத்தைக் குந்தான்
நாவறளும் வாயுவது மீறுந்தானே"

– சித்த மருத்துவம்

Tongue generally becomes dry.

Abdomen is distended due to flatulence.

அவத்தை – 4

"தானான நாலவத்தை யங்க தாகம்
சன்னியது பாத முண்டாம்"

– சித்த மருத்துவம்

Severe thirst occurs.

Causes Delirium.

அவத்தை – 5

‘‘ஐந்து வத்தைத்
தேறான நீர்பெருகந் தாது நஷ்டம்’’

– சித்த மருத்துவம்

Quantity of urine increased.

Loss of semen (impotence).

அவத்தை – 6

‘‘ நிலை யாறா மவத்தையுடற் கிடை கொள்ளாது
மூனான மூர்ச்சை வரும்’’

– சித்த மருத்துவம்

Sleeplessness is present.

Difficulty in breathing is experienced.

அவத்தை – 7

‘‘ஏழுவத்தை
மிக்கவரோ சிகஞ்சுவாசந் தேக சாட்யம்’’

– சித்த மருத்துவம்

Tongue becomes tasteless.

Difficulty in breathing is experienced.

Loss of Appetite.

General weakness persists.

அவத்தை – 8

‘‘ஏனான எட்டாவ தவத்தை தானே
எழுகிரந்தி பிளவையுந்தான் மிகவுண்டாமே’’

– சித்த மருத்துவம்

Abscess is formed.

Presence of Carbuncle

அவத்தை – 9

‘‘உண்டாகு மொன்பதா மவத்தை கேளாய்
ஒழுக்கான ஆசாரங் கிருமி யுண்டாம்’’

– சித்த மருத்துவம்

Irregularities in daily habits like bowel habits.

Recurrent Infection occurs.

அவத்தை – 10

"பண்டான பத்தாந்த வைத்தைக் கேளாய்
பாரமாம் சயங்கண்டு பரத்துக்கேகும்"

– சித்த மருத்துவம்

Secondary infection like Tuberculosis may sets in due to loss of immunity.

Other complications of Madhumegam:

Meganeer Kattigal (Diabetic Carbuncle)

1. Madaku Katti
2. Ammaiodu Katti
3. Valai Kann Katti
4. Athomuga Katti
5. Paisura Katti
6. Kadalai Katti
7. Kadugu Katti
8. Thirathi Katti
9. Nilapoosani Katti
10. Megavithirathi Katti

PROGNOSIS (தீரும் தீராதவை):

"செய்யவே வச்சரமாந் தண்ட மான
செயமான முதுகுதண்டைப் பற்றி நிற்கும்
பெய்யவே பெருநரம்பில் மேகந்தானும்
பிறக்குமென்றே தானறி ந்து வாதந்தன்னால்
பிய்யவே பிறந்தகல மாறா லசாத்தியம்
பித்தத்திற் பிறந்தசல மாறும் யாப்யம்
பையவே சேட்டுமத்திற் பிறந்த பத்தும்
பரமனுரைத் தார் சாத்யம் பராபரிக்கே"

– யூகி வைத்திய சிந்தாமணி [42]

"வழியும் வாதம் நான்காமே மாறா தவிழ்தந் தன்னாலே
பொழியும் வாதம் நில்லாது போமே மருந்தைப் பொய்யெனவே"

– தேரையர் வாகடம் [43]

The four types of Megam caused as a result of imbalance of Vali are incurable.
The six types of Megam arising with disparity of Azhal could be cured with great difficulty.
But ten types of megam arising due to Iyyam are curable.

எந்தெந்த ரோகங்களில் சிறுநீர் அதிகரித்தாலும் குறைந்தாலும் தீது

"வெப்பு பிணியதனில் வெம்மேகத்தால் வருந்தின்
தப்பு மிகை நீரே தானிறங்கின் – செப்பும்
கிராணியிற் பாண்டில் குளர்நீர் சுருங்கிற்
பிராணன் பிரியுமெனப் பேசு"

– கண்ணு சாமியம் [44]

Very excess of urination in Megaroham causes death.

"துதிப்பான மேகத்தில் நீரிழிவு மாகா
தோன்றிய நீரிழிவு தண்ணீர் வாதமுமாகா"

–சதக நாடி [45]

If Megaroham is associated with excessive urination, it is difficult to cure. If Megaroham coexist with vali it is incurable.

"மேகத்தில் நீரிழிவு மேவுமதில் வாத நோய்
வேக வயித்துள் வயிற்றுளைவு – சோக விக்கல் பன்னு விக்கில்
தன்னில் பகரிளைப்புப் பாங்க தனிற்
பின்னளை யாகாது பேசு"

– நோய் நாடல். [46]

In Megaroham complications associated with carbuncle, Morbid thirst, excessive body heat, shock and sweat occurs and the prognosis will be bad.

"வேர்வைதனிற் கபமும் மேவுமதில் விக்கல் நோய்
கார்முகில் நேர் கூந்தலாய் கண்டு மேல் – சீர்கொள்
மருத்துவத்திற்றேர்ந்த மதியுடையாராவி
தரித்திரா தென்பர்சரி"

– நோய் நாடல் [47]

If Iya megam is associated with sweat hiccough, the prognosis is bad.

மேகம் இருபதுக்கும் பத்தியம்:

"மேக மிருபதுக்கு பத்தி யந்தான்
பாங்காக வுரைத்திடவே பசுவின் வெண்ணெய்
பொரிவாக எருமை மோர் பொன்னாங்காணி
பேர்பெற்ற சிறுகீரை முசுட்டை யாகும்
ஆரிவாக அவரையொடு புடல் முருங்கை
அதிசரமாங் கண்டு சருக் கரையு மாகும்
மரிவாக மாதளையாம் பேரீத் தாகும்
மகாவிளம் பழமுந்திரிப் பழமு மாமே
ஆமேபே யன் வாழைப் பழமுங் கச்சல்
அத்தியிடப் பிஞ்சுசிறு பயறுபழஞ் சோறு
பாமேபழஞ் சோற்றுநீர் வெந்தயஞ் சீரகமாம்
பாகல் பீரிக் கங்காய்கரு வேப்பிலை கொத்தமலி
நேமேநெற் பொறி எள்ளு முசுமுசுக்கை யாகும்
நேரான நல்லெண்ணெய் புண்ணக்குளுந்து
தாமேக மிருபதுக்கும் பத்ய வர்க்கம்
சாற்றினார் சிவன்றானுந் தாய்க்குத் தானே"

– யூகி வைத்திய சிந்தாமணி [48]

The following foods are preferred for Madhumegam:

பொன்னாங்கண்ணி, சிறுகீரை, கொத்தமல்லி, கருவேப்பிலை, முசுமுசுக்கை, பாகல், பீர்க்கு, அவரை, முருங்கை, வெந்தயம், சீரகம், எள், பசுவின் வெண்ணெய், எருமை மோர், வெந்தயம், சிறுபயிறு, உளுந்து இவை மேகம் இருபதுக்கும் பத்தியம் ஆகும்.

MARUTHUVAM:

The treatment in Siddha Medicine is aimed at keeping the three kutrams (Vali, Azhal and Iyam) in equilibrium and maintenance of the seven udal thathukal.

In Siddha science, the treatment is not only for removal of the disease, but for the prevention and improving the body condition after the removal of the disease.

This is classified as Kappu (Prevention), Neekam (Treatment) and Niraivu (Restoration)

- **Kappu:**

  Prevention is better than cure is a proverb.

  Siddha principles based mainly on prevention as mentioned in "Theraiyar pini Annuga vithi" by Theraiyar. The aim of the treatment is to bring the affected thathus and Mukkutram to normal levels by eyamma, niyamma, diet and medicine.

- **Neekam:**

  For the disease Madhumegam, PUNGAMPOO CHOORNAM - 2gm twice a day is given.

- **Niraivu:**

  Physical, Psychological, social and economic rehabilitation of individual is known as Niraivu.

  In Madhumegam, Azhal kutram and other two kutrams Vali and Iyam deranged and causes impairment of dasavayu which in turn affect the seven udal thathukkal.

PHYSICAL EXERCISES

"நண்பு பெற வுண்ட பின்பு குறுநடையுங் கொள்வோம்"

– பதார்த்த குண சிந்தாமணி [49]

The need for walking is emphasized by Therayar. Exercise forms an important component along with drugs and diet management of Diabetes Mellitus – Type II. Patients should be encouraged to take regular physical activity in form of walking, gardening, swimming and cycling for 30 minutes daily to improve insulin sensitivity and lipid profile and lower blood pressure.

YOGA FOR MADHUMEGAM

YOGA:

Yogic Physical exercise makes the muscles healthy and strong. It also tones up all the involuntary organs which are concerned digestion, evacuation, circulation, respiration and secretion and through them, the autonomic nervous system which regulates their activities.

- Yoga Aganas for Health & Vigour [50]

Yoga is primarily the process of self culture. Yoga according to Thirumandiram is the attainment of spiritual, psychological and physical perfection.

"உடம்பா லழியில் உயிராலழிவர்
திடம்பட மெய்ஞானம் சேரவும் மாட்டார்
உடம்பை வளர்க்கும் உபாயம் அறிந்தே
உடம்பை வளர்தேன் உயிர் வளர்தேனே"

- திருமூலர் [51]

As a body is said to be divine, Thirumoolar advised that each and every individual aspiring for self realisation should practice yoga. It is a science that helps to lead a pure and healthy life, the practice of yoga prevents the decay of tissues by increasing with abundant energy force.

"இயமம் நியமம் என்னிலாஆதனம்
நயமுறு பிரணாயாமம் பிரத்தியாகாரம்
சயமுறு தாரனை தியானம் சமாதி
அயமுறு அட்டாங்க மாவது மாமே"

- திருமூலர் [52]

Asanas are nothing but a kind of yoga. There are innumerable types asanaas described in Siddha text. Each and every yogasana is indicated for a definite effect in a particular region of the body by stimulating the internal organs to function in a normal way.

The following Asanaas will help to manage madhumegam:

1. Mayurasanam
2. Dhanurasanam
3. Paschimottanasanam
4. Pawanamukthasanam
5. Ardha Matsyendrasanam

1. Mayurasanam:

Mayur means peacock. In this asanam the legs are stretched like the wings of the peocock and so get the name Mayurasanam. The palms on the floor, and put pressure on both sides of the elbow with the stomach, the whole body without touching the ground, like an elevated position that is called mayurasanam. Given below is a step by step procedure to perform and benefits of this asanam.

Practice:

- First kneel down on the floor, then make sure that there is some space between the legs.
- Now join the hands and keep the palms in between the gap of the legs.
- Then bend a little and keep the abdomen on the elbow of the hands.
- Stretch the legs one by one slowly and straighten the elbows.
- The toes alone should be on the ground.
- Looking at this now the head to legs the body should be on a straight line.

- After being at the same stage we have to be there for some time and then release the breath slowly and hold all your weight on the elbow. Now bend your head little towards front and rise your legs a little slanting.
- Be at the same position for 20 to 30 seconds. Then slowly kneel down your legs and release the hands.

Benefits:

It induces the pancreas and helps in preventing diabetes.

2. Dhanurasanam:

Dhanurasanam has been named after the shape the body takes while performing it - that of a bow. Dhanu means bow.

Practice:

1. Lie on your stomach with your feet hip-width apart and your arms by the side of your body.
2. Fold your knees, take your hands backwards and hold your ankles.
3. Breathing in, lift your chest off the ground and pull your legs up and back.
4. Look straight ahead with a smile on your face.
5. Keep the pose stable while paying attention to your breath. Your body is now curved and taut as a bow.
6. Continue to take long deep breaths as you relax in this pose. But bend only as far as your body permits you to. Do not overdo the stretch.
7. After 15 -20 seconds, as you exhale, gently bring your legs and chest to the ground. Release the ankles and relax.

Benefits:

Helps regulate the pancreas and is recommended for people with diabetes.

3. Paschimottanasanam

"**Paschima**" means your "**back**" and "**Uttana**" means "**stretching**". This asana covers the stretching of the whole body from head to heels so it is called as Paschimottanasnam.

Practice:

1. Sit down straight with your legs together by stretching in front of you. keep your head neck and spine erect.
2. Place the palms on your respective knees.
3. Now bend your head and trunk slowly forward to catch the toes with the thumb, index and middle fingers without bending knees.
4. Take a deep breath and exhale slowly. Try to touch your head to your both knees as shown in above image.
5. Bend the arm and try to touch the elbow on the floor.
6. Exhale completely and holding out your breath stay in this posture for a few seconds.
7. After few seconds slowly return to your starting position.
8. Breathe normally.
9. Repeat this for 3-4 times.

Benefits:

1. It acts as a stress reliever.
2. Reduces fatty deposits in the abdomen.
3. Remove anxiety, anger and irritability.
4. It induces the pancreas and helps in preventing diabetes.

4. Pawana muktasanam:

Pawana muktasanam knows as Wind Removing Pose. It is beneficial to cure gas problems and poor digestion. Regular practice of Pawanamuktasanam helps to stimulate bowel movement which is very necessary for removing waste material.

Practice:

1. Lie flat on your back and keep the legs straight and relax breathe deeply and rhythmically.
2. Inhale slowly and lift the legs and bend in the knee. Bring upwards to the chest till your thigh touches to stomach
3. Hug your knees in place and lock your fingers.
4. Try to touch the knee with your nose tip. This is not easy in first time. But regular practice you can do this. Hold this position for 20 to 30 seconds. You can extend it till 1 minute as per your capability.
5. Now exhale slowly and come back to the original position that is Shavasana (Lie straight)
6. This is very beneficial for stomach abs. The results are very impressive.
7. Practice 3 to 5 cycles each day.

Benefits:

1. Cures acidity Indigestion and Constipation.
2. Very good for all abdominal organs.
3. Regular practice cures gastrointestinal problems.
4. It induces the pancreas and helps in preventing diabetes.

5. Matsyendrasanam:

Ardha means Half, Matsyendra means King of fish.

Practice:

- Sit with the legs straight and relax the whole body.
- Place the sole of the right foot flat on the floor on the outside of the left knee.
- Bend the left leg and lay the left heel beside the right buttock. Both buttocks remain on the floor. The back is upright and relaxed.
- Bring the left arm to the outside of the right knee and grasp the right ankle.
- Turn the upper body as far as possible to the right, place the right arm across the back and look over the right shoulder. Breathing normally remains for a few minutes in this position and relax the whole body.
- Slowly return to the starting position.

Benefits:

Promotes mobility of the spine and hips. The twist aids release of tension from the deep layers of muscle in the back. The breath is also deepened in this position. Function of the kidneys and pancreas is stimulated and the ability to concentrate is improved. [53]

MODERN ASPECTS

MODERN ASPECTS

DEFINITION

Diabetes Mellitus is a group of metabolic disorder characterized by Hyperglycaemia resulting from defects in Insulin secretion, Insulin action, or both. The chronic Hyperglycaemia of Diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels.

Several pathogenic processes are involved in the development of Diabetes. These range from autoimmune destruction of the β-cells of the Pancreas with consequent Insulin deficiency to abnormalities that result in resistance to Insulin action. The basis of the abnormalities in carbohydrate, Fat, and Proteins metabolism in Diabetes is deficient action of Insulin on target tissues. Deficient Insulin action results from inadequate Insulin secretion and/or diminished tissue responses to Insulin at one or more points in the complex pathways of hormone action. Impairment of Insulin secretion and defects in Insulin action frequently coexist in the same patient, and it is often unclear which abnormality, if either alone, is the primary cause of the Hyperglycaemia. [54]

EPIDEMIOLOGY:

The application of Epidemiology to the study of Diabetes Mellitus has provided valuable information on several aspects of this disease such as its natural history, prevalence, incidence, morbidity and mortality in diverse populations around the world. Identification of the cause of the disease and the possible preventive measures that could be instituted to arrest or delay the onset of this disease which has reached epidemic proportions in both the developed and the developing nations. Unfortunately, the improvement in outcomes for individual patients with Diabetes has not resulted in similar improvements from the public health perspective. [55]

Diabetes is fast gaining the status of a potential Epidemic in India with more than 62 million Diabetic individuals currently diagnosed with the disease. In 2000, India (31.7 million) topped the world with the highest number of people with Diabetes Mellitus followed by China (20.8 million) and United States (17.7 million). The prevalence of Diabetes is predicted to double globally from 171 million in 2000 to 366 million in 2030 with a maximum increase in India. It is predicted that, by 2030 Diabetes Mellitus may afflict up to 79.4 million individuals in India, while China

(42.3 million) and the United States (30.3 million) will also see significant increase in those affected by the disorder. [56]

India currently faces an uncertain future in relation to the potential burden that Diabetes may impose upon the country. Many influences affect the prevalence of disease throughout a country, and identification of those factors is necessary to facilitate change when facing health challenges. [57]

ETIOLOGY:

Type I Diabetes occurs when your immune system, the body's system for fighting infection, attacks and destroys the Insulin-producing beta cells of the Pancreas. Type I Diabetes is caused by genes and environmental factors, such as viruses, that might trigger the disorder.

Type II Diabetes is the most common form of Diabetes and it is caused by several factors, including lifestyle factors and genes.

Gestational Diabetes is a type of Diabetes that develops during pregnancy, and is caused by the hormonal changes of pregnancy along with genetic and lifestyle factors. [58]

OTHER CAUSES:

❖ GENETIC MUTATIONS:

- Monogenic Diabetes is caused by mutations, or changes, in a single gene. These changes are usually passed through families, but sometimes the gene mutation happens on its own. Most of these gene mutations cause Diabetes by making the Pancreas less able to make Insulin. The most common types of monogenic Diabetes are Neonatal Diabetes and Maturity-Onset Diabetes of the Young (MODY). Neonatal Diabetes occurs in the first 6 months of life.
- Cystic Fibrosis produces thick mucus that causes scarring in the Pancreas. This scarring can prevent the Pancreas from making enough Insulin.
- Hemochromatosis causes the body to store too much iron. If the disease is not treated, iron can build up in and damage the Pancreas and other organs.

❖ HORMONAL DISEASES:

Some hormonal diseases cause the body to produce too much of certain hormones, which sometimes cause Insulin resistance and Diabetes.

- Cushing's syndrome occurs when the body produces too much Cortisol and often called the "Stress hormone."
- Acromegaly occurs when the body produces too much growth hormone.
- Hyperthyroidism occurs when the thyroid gland produces too much thyroid hormone.

❖ DAMAGE OR REMOVAL OF THE PANCREAS:

Pancreatitis, pancreatic cancer, and trauma can all harm the beta cells or make them less able to produce Insulin, resulting in Diabetes. If the damaged Pancreas is removed, Diabetes will occur due to the loss of the beta cells. [59]

PATHOPHYSIOLOGY:

There is a direct link between Hyperglycaemia and physiological and behavioural responses. Whenever there is Hyperglycaemia, the brain recognizes it and sends a message through nerve impulses to Pancreas and other organs to decrease its effect.

- TYPE I DIABETES MELLITUS:

Type I Diabetes is characterized by autoimmune destruction of Insulin producing cells in the Pancreas by CD4+ and CD8+ T cells and macrophages infiltrating the Islets. Several features characterize Type I Diabetes Mellitus as an autoimmune disease.

1. Presence of immuno-competent and accessory cells in infiltrated Pancreatic Islets.
2. Association of susceptibility to disease with the class II (immune response) genes of the major histocompatibility complex (MHC; Human Leucocyte Antigens HLA).
3. Presence of Islet cell specific autoantibodies.
4. Alterations of T cell mediated immune regulation, particularly in CD4+ T cell compartment.

5. The involvement of monokines and TH1 cells produce inginterleukins in the disease process.
6. Response to immunotherapy and
7. Frequent occurrence of other organ specific auto- immune diseases in affected individuals or in their family members.

Approximately 85% of patients have circulating islet cell antibodies, and the majorities also have detectable anti-Insulin antibodies before receiving Insulin therapy. Most Islet cell antibodies are directed against Glutamic Acid Decarboxylase (GAD) within pancreatic β cells.

- TYPE II DIABETESMELLITUS:

The two main pathological defects in Type II Diabetes are impaired Insulin secretion through a dysfunction of the pancreatic β-cell, and impaired Insulin action through Insulin resistance. In situations where resistance to Insulin predominates, the mass of β-cells undergoes a transformation capable of increasing the Insulin supply and compensating for the excessive and anomalous demand.

In absolute terms, the plasma Insulin concentration (both fasting and meal stimulated) usually is increased but although relative to the severity of Insulin resistance, the plasma Insulin concentration is insufficient to maintain normal Glucose Homeostasis. Keeping in mind the intimate relationship between the secretion of Insulin and the sensitivity of hormone action in the complicated control of Glucose Homeostasis, it is practically impossible to separate the contribution of each to the etiopathogenesis of Diabetes Mellitus type II.

Insulin resistance and HyperInsulinemia eventually lead to Impaired Glucose Tolerance. Except for Maturity Onset Diabetes of the Young(MODY), the mode of inheritance for Type II Diabetes Mellitus is unclear. MODY, inherited as an autosomal dominant trait, may result from mutations in Glucokinase gene on chromosome 7p. MODY is defined as Hyperglycaemia diagnosed before the age of twenty-five years and treatable for over five years without Insulin in cases where Islet cell antibodies (ICA) are negative.

- INSULIN RESISTANCE:

The primary events are believed to be an initial deficit in Insulin secretion and in many patients relative Insulin deficiency in association with peripheral Insulin resistance. Resistance to the action of Insulin will result in impaired Insulin mediated glucose uptake in the periphery (by muscle and fat), incomplete suppression of hepatic glucose output and impaired triglyceride uptake by fat. To overcome the Insulin resistance, Islet cells will increase the amount of Insulin secreted. Endogenous glucose production is accelerated in patients with Type II Diabetes or Impaired Fasting Glucose. Because this increase occurs in the presence of Hyper Insulinemia, at least in the early and intermediate disease stages, hepatic Insulin resistance is the driving force of Hyperglycaemia of Type II Diabetes. [60]

THE PANCREAS:

The Pancreas is an Abdominal Glandular organ, with a Digestive (Exocrine) and Hormonal (Endocrine) function.

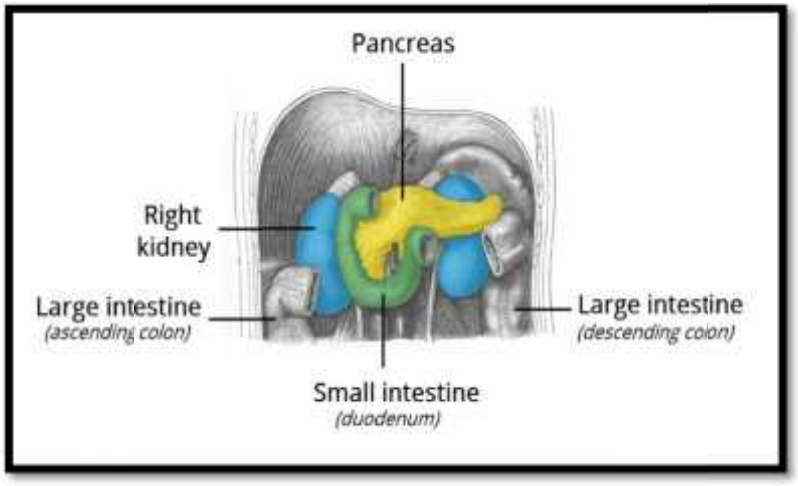
- ANATOMICAL POSITION:

The Pancreas is an oblong-shaped and flattened organ, about the size of a hand. Aside from the tail, it is a Retroperitoneal structure (lies behind the peritoneal cavity), located deep within the upper abdomen in the epigastrium and left hypochodrium regions.

Within the abdomen, the Pancreas is surrounded by other viscera and vessels.

- Stomach – lies Anteriorly and Superiorly.
- Duodenum – situated Anteriorly and Medially, curving around the head of the Pancreas.
- Spleen – located Posteriorly and Laterally. It is connected by ligaments to the tail of the Pancreas.
- Vasculature – the Aorta and Inferior vena cava pass Posteriorly to the head of the Pancreas.

ANTERIOR VIEW OF ABDOMEN



- ANATOMICAL STRUCTURE:

The Pancreas is typically divided into five parts:

1. Head:

   This is the widest part of the Pancreas. It lies within the C-shaped curve created by the Duodenum, and is connected to it by connective tissue.

2. Uncinate process:

   This is a projection arising from the lower part of the head and extending medially to lie beneath the body of the Pancreas. It lies posterior to the superior mesenteric vessels.

3. Neck:

   Located between the head and the body of the Pancreas. It overlies the superior Mesenteric vessels which form a groove in its posterior aspect.

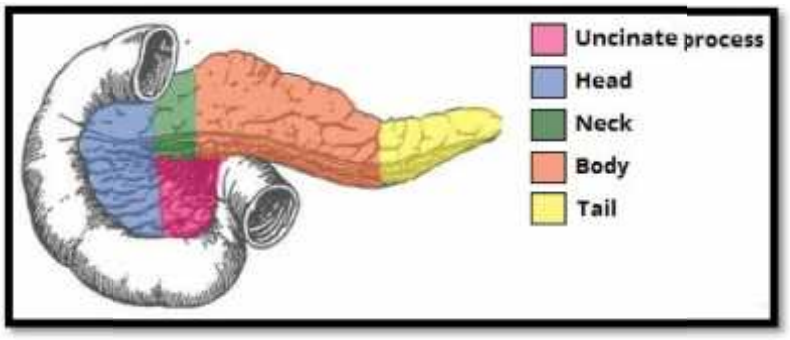
4. Body:

   The body is centrally located, crossing the midline of the human body to lie behind the Stomach and to the left of the Superior Mesenteric vessels.

5. Tail:

   The left end of the Pancreas that lies within close proximity to the hilum of the Spleen. It is contained within the Spleno -renal ligament with the Splenic vessels. This is the only part of the Pancreas that is intraperitoneal.

PARTS OF THE PANCREAS



- THE DUCT SYSTEM:

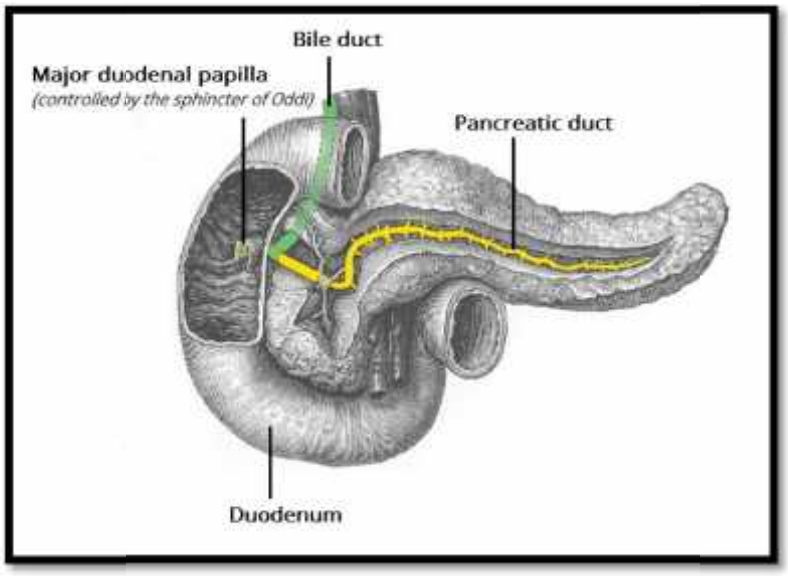
The Exocrine compartment is classified as a Serous gland. It is composed of approximately a million 'Berry-like' clusters of cells called Acini, connected by short intercalated ducts.

Intercalated duct cells beginning within Acini are called Centroacinar cells. The intercalated ducts drain into a network of intralobular collecting ducts, which in turn drain into the main Pancreatic duct.

The Pancreatic duct runs the length of the Pancreas and unites with the common bile duct, forming the Hepato-pancreatic Ampulla of Vater. This structure opens into the Duodenum.

Secretions into the Duodenum are controlled by a muscular valve, the sphincter of Oddi. It surrounds the Ampulla of Vater, acting as a valve.

EXOCRINE PANCREAS SECRETING INTO THE DUODENUM

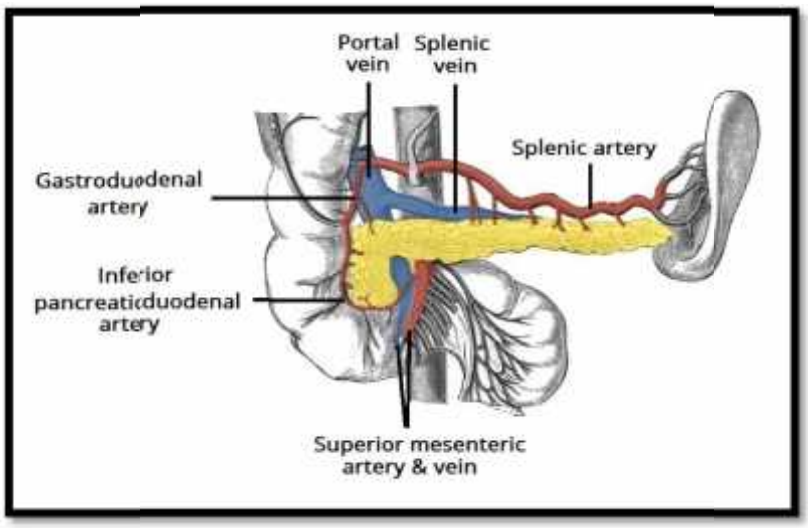


- VASCULATURE:

The Pancreas is supplied by the Pancreatic branches of the Splenic artery. The head is additionally supplied by the superior and inferior Pancreatico-duodenal arteries which are branches of the Gastro-duodenal and superior Mesenteric arteries, respectively.

Venous drainage of the head of the Pancreas is into the superior Mesenteric branches of the Hepatic portal vein. The Pancreatic veins draining the rest of the Pancreas do so into the Splenic vein.

THE ARTERIAL SUPPLY AND VENOUS DRAINAGE OF THE PANCREAS



- LYMPHATICS:

The Pancreas is drained by lymphatic vessels that follow the arterial supply. They empty into the Pancreatico- Splenal nodes and the pyloric nodes, which in turn drain into the superior Mesenteric and Celiac lymph nodes.

- CELLS AND SECRETIONS OF THE PANCREATIC ISLETS:

The Pancreatic Islets each contain four varieties of cells:

1. The alpha cell produces the hormone Glucagon and makes up approximately 20 percent of each Islet. Glucagon plays an important role in blood Glucose regulation, low blood glucose levels stimulate its release.
2. The beta cell produces the hormone Insulin and makes up approximately 75 percent of each Islet. Elevated blood Glucose levels stimulate the release of Insulin.
3. The delta cell accounts for four percent of the Islet cells and secretes the peptide hormone Somatostatin. It is also released by the hypothalamus (as GHIH). Pancreatic Somatostatin inhibits the release of both Glucagon and Insulin.
4. The Pancreatic polypeptide cell accounts for about one percent of Islet cells and secretes the pancreatic polypeptide hormone. It is thought to play a role in appetite, as well as in the regulation of Pancreatic Exocrine and Endocrine secretions. Pancreatic polypeptide released following a meal may reduce further food consumption, however, it is also released in response to fasting.

- REGULATION OF BLOOD GLUCOSE LEVELS BY INSULIN AND GLUCAGON:

  Glucose is required for cellular respiration and is the preferred fuel for all body cells. The body derives glucose from the breakdown of the carbohydrate-containing foods and drinks. Glucose not immediately taken up by cells for fuel can be stored by the liver and muscles as Glycogen, or converted to Triglycerides and stored in the adipose tissue. Hormones regulate both the storage and the utilization of glucose as required. Receptors located in the Pancreas sense blood glucose levels, and subsequently the pancreatic cells secrete Glucagon or Insulin to maintain normal levels.

- GLUCAGON:
  - Receptors in the Pancreas can sense the decline in blood glucose levels, such as during periods of fasting or during prolonged labour or exercise. In response, the alpha cells of the Pancreas secrete the hormone Glucagon, which has several effects:
  - It stimulates the liver to convert its stores of glycogen back into glucose. This response is known as Glycogenolysis. The glucose is then released into the circulation for use by body cells.
  - It stimulates the liver to take up amino acids from the blood and convert them into glucose. This response is known as Gluconeogenesis.
  - It stimulates Lipolysis, the breakdown of stored Triglycerides into free Fatty acids and Glycerol. Some of the free glycerol released into the bloodstream travels to the liver, which converts it into glucose. This is also a form of Gluconeogenesis.
  - These actions increase blood glucose levels. The activity of Glucagon is regulated through a Negative feedback mechanism, rising blood glucose levels inhibit further Glucagon production and secretion.

- INSULIN:

The primary function of Insulin is to facilitate the uptake of glucose into body cells. Red blood cells, as well as cells of the brain, liver, kidneys, and the lining of the small intestine, do not have Insulin receptors on their cell membranes and do not require Insulin for glucose uptake. Although all other body cells do require Insulin if they are to take glucose from the bloodstream, skeletal muscle cells and adipose cells are the primary targets of Insulin.

The presence of food in the intestine triggers the release of gastrointestinal tract hormones such as glucose-dependent insulinotropic peptide (previously known as Gastric inhibitory peptide). This is in turn the initial trigger for Insulin production and secretion by the beta cells of the Pancreas. Once nutrient absorption occurs, the resulting surge in blood glucose levels further stimulates Insulin secretion.

Precisely how Insulin facilitates glucose uptake is not entirely clear. However, insulin appears to activate a Tyrosine kinase receptor, triggering the phosphorylation of many substrates within the cell. These multiple biochemical reactions converge to support the movement of intracellular vesicles containing facilitative glucose transporters to the cell membrane. In the absence of Insulin, these transport proteins are normally recycled slowly between the cell membrane and cell interior. Insulin triggers the rapid movement of a pool of glucose transporter vesicles to the cell membrane, where they fuse and expose the glucose transporters to the extracellular fluid. The transporters then move glucose by facilitated diffusion into the cell interior. [61]

CLASSIFICATION

The new classification is primarily based on etiologies. The staging of patho-physiology by degree of deficiency of insulin is also adopted. The previous terms, insulin-dependent diabetes mellitus (IDDM) and non-insulin dependent diabetes mellitus (NIDDM), are abandoned. Instead, the terms type 1 and type 2 diabetes mellitus are used for etiological classification. The etiologic classifications of Diabetes Mellitus are listed below.

TYPE I DIABETES:

Type I Diabetes Mellitus (Juvenile Diabetes) is characterized by betacell destruction caused by an autoimmune process, usually leading to absolute Insulin deficiency. Type I is usually characterized by the presence of anti–glutamic acid decarboxylase, Islet cell or Insulin antibodies which identify the autoimmune processes that lead to betacell destruction.

- IMMUNE-MEDIATED DIABETES:

This form of Diabetes, which accounts for only 5–10% of those with Diabetes, previously encompassed by the terms Insulin-Dependent Diabetes. Type I Diabetes or Juvenile-onset Diabetes, results from a cellular-mediated autoimmune destruction of the β-cells of the Pancreas.

In this form of Diabetes, the rate of β-cell destruction is quite variable, being rapid in some individuals (mainly infants and children) and slow in others (mainly adults). Some patients, particularly children and adolescents, may present with ketoacidosis as the first manifestation of the disease. Others have modest fasting hyperglycaemia that can rapidly change to severe Hyperglycaemia and/or ketoacidosis in the presence of infection or other stress.

- IDIOPATHIC DIABETES:

Some forms of Type I Diabetes have no known etiologies. Some of these patients have permanent insulinopenia and are prone to ketoacidosis, but have no evidence of autoimmunity. Although only a minority of patients with Type I Diabetes fall into this category most are of African or Asian ancestry. Individuals with this form of Diabetes suffer from episodic Ketoacidosis and exhibit varying degrees of insulin deficiency between episodes.

TYPE II DIABETES MELLITUS:

This form of Diabetes, which accounts for approximately 90–95% of those with Diabetes, previously referred to as Non–Insulin-Dependent Diabetes, Type II Diabetes, or Adult-Onset Diabetes, encompasses individuals who have Insulin resistance and usually have relative (rather than absolute) insulin deficiency.

Most individuals with Type II Diabetes exhibit intra-abdominal (visceral) obesity, which is closely related to the presence of Insulin resistance. In addition, hypertension and dyslipidemia (high triglyceride and low HDL-cholesterol levels, postprandial hyperlipidemia) often are present in these individuals.

This is the most common form of Diabetes Mellitus and is highly associated with a family history of Diabetes, older age, obesity and lack of exercise. It is more common in women, especially women with a history of Gestational Diabetes, and in blacks, hispanics and native americans.

GESTATIONAL DIABETES MELLITUS (GDM):

Gestational Diabetes Mellitus is an operational classification (rather than a pathophysiologic condition) identifying women who develop Diabetes Mellitus during gestation. Women who develop Type I Diabetes Mellitus during pregnancy and women with undiagnosed asymptomatic Type II Diabetes Mellitus that is discovered during pregnancy are classified with Gestational Diabetes Mellitus (GDM). In most women who develop GDM, the disorder has its onset in the third trimester of pregnancy.

OTHER SPECIFIC TYPES:

- MONOGENIC DIABETES:

Types of Diabetes Mellitus of various known etiologies are grouped together to form the classification called "Other Specific Types". This group includes persons with genetic defects of beta-cell function (this type of Diabetes was formerly called MODY or Maturity-onset Diabetes in youth) or with defects of insulin action.

Persons with dysfunction associated with other endocrinopathies (e.g. Acromegaly), and persons with pancreatic dysfunction caused by drugs, chemicals or infections and they comprise less than 10% of DM cases. [62]

RISK FACTORS:

The risk factors for Type I Diabetes are still being researched. However, having a family member with Type I Diabetes slightly increases the risk of developing the disease. Environmental factors and exposure to some viral infections have also been linked to the risk of developing Type I Diabetes.

Several risk factors have been associated with Type II Diabetes and include:

- Family history of Diabetes
- Overweight
- Unhealthy diet
- Physical inactivity
- Increasing age
- High Blood pressure
- Ethnicity
- History of Gestational Diabetes
- Poor nutrition during pregnancy
- Impaired Glucose Tolerance (IGT) is a category of higher than normal blood glucose, but below the threshold for diagnosing Diabetes.
- Changes in diet and physical activity related to rapid development and urbanisation have led to sharp increases in the numbers of people developing Diabetes.
- Pregnant women who are overweight, have been diagnosed with IGT, or have a family history of Diabetes are all at increased risk of developing Gestational Diabetes Mellitus (GDM). In addition, having been previously diagnosed with Gestational Diabetes or being of certain ethnic groups puts women at increased risk of developing GDM. [63]

CLINICAL FEATURES:

Most of the symptoms are similar in both types of Diabetes but they vary in their degree and develop more rapidly in Type I Diabetes and more typical. Some of the clinical features and symptoms are listed below.

- Glucosuria:

  When blood glucose level is above 180mg/dl, glucose appears in urine. It is the renal threshold for glucose.

- Osmotic Diuresis:

  The excess glucose in renal tubules decreases reabsorbtion of water result in diuresis. This leads to polyuria, polydipsia.

- Polyuria:

  The amount of urine may be several litres in 24 hours. This is due to excessive sugar in the urine which acts as a Diuretic.

- Polydipsia, Dryness of mouth:

  Polyuria decreases water content in the body stimulates taste centre and in turn increases water intake.

- Polyphagia and Predilection for sweet food:

  This symptom is due to non utilization of sugar for energy expenditure.

- Asthenia:

  This is due to proteins depletion and increased utilization of proteins for energy.

- Emaciation:

  It is due to loss of water, glycogen and triglyceride and proteins stores and gradually reduced muscle mass occurs.

- Pruritis vulvae ,Balanitis, Genital Candidiasis, Skin Sepsis (Boils):

  This is due to irritant action of sugar on the tissue and fungal or bacterial infections.

- Constipation:

  The stool becomes hard and bowel movement may take place after every 2 to 3 days.

- Nausea, Headache, Blurring of Vision,
- Mood change, Irritability, Difficulty in concentrating, Apathy, Unhealed wounds.
- Frequent changes in Refractive error and Cataract. [64]

COMPLICATIONS:

People with Diabetes have an increased risk of developing a number of serious health problems. Consistently high blood glucose levels can lead to serious diseases affecting the heart and blood vessels, eyes, kidneys, nerves and teeth. In addition, people with Diabetes also have a higher risk of developing infections. In almost all high-income countries, Diabetes is a leading cause of cardiovascular disease, blindness, kidney failure, andlower limb amputation.

Maintaining blood glucose levels, blood pressure, and cholesterol at or close to normal can help delay or prevent Diabetes complications. Therefore people with Diabetes need regular monitoring.

- CARDIOVASCULAR DISEASE:

Affects the heart and blood vessels and may cause fatal complications such as Coronary Artery Disease (leading to heart attack) and Stroke. Cardiovascular Disease is the most common cause of death in people with Diabetes. High blood pressure, high cholesterol, high blood glucose and other risk factors contribute to increasing the risk of cardiovascular complications.

- DIABETIC NEPHROPATHY:

This is caused by damage to small blood vessels in the kidney's leading to becoming less efficient or to fail altogether. Kidney disease is much more common in people with Diabetes than in those without Diabetes. Maintaining near normal levels of blood glucose and blood pressure can greatly reduce the risk of kidney disease.

- DIABETIC NEUROPATHY:

Diabetes can cause damage to the nerves throughout the body when blood glucose and blood pressure are too high. This can lead to problems with digestion, erectile dysfunction, and many other functions. Among the most commonly affected areas are the extremities, in particular the feet. Nerve damage in these areas is called Peripheral Neuropathy, and can lead to pain, tingling, and loss of feeling. Loss of feeling is particularly important because it can allow injuries to go unnoticed, leading to serious infections and possible amputations. People with Diabetes carry a risk of amputation that may be more than 25 times greater than that of people without Diabetes. However, with comprehensive management, a large proportion of amputations related to Diabetes can be prevented. Even when amputation takes place, the remaining leg and the person's life can be saved by good follow-up care from a multidisciplinary foot team. People with Diabetes should regularly examine their feet.

- DIABETIC RETINOPATHY:

Most people with Diabetes will develop some form of Eye disease (Retinopathy) causing reduced vision or blindness. Consistently high levels of blood glucose, together with high blood pressure and high cholesterol, are the main causes of Retinopathy. It can be managed through regular eye checks and keeping glucose and lipid levels at or close to normal.

- FOOT DAMAGE:

Nerve damage in the feet or poor blood flow to the feet increases the risk of various foot complications. Left untreated, cuts and blisters can develop serious infections, which often heal poorly. These infections may ultimately require toe, foot or leg amputation.

- SKIN CONDITIONS:

Diabetes may leave you more susceptible to skin problems, including bacterial and fungal infections.

- HEARING IMPAIRMENT:

Hearing problems are more common in people with Diabetes.

- ALZHEIMER'S DISEASE:

Type II Diabetes may increase the risk of Alzheimer's disease. The poorer your blood sugar control, the greater the risk appears.

- COMPLICATIONS OF GESTATIONAL DIABETES:

Most women who have Gestational Diabetes deliver healthy babies. However, Untreated or Uncontrolled blood sugar levels can cause problems for mother and baby.

I. PREGNANCY COMPLICATIONS:

Women with any Type of Diabetes during pregnancy risk a number of complications if they do not carefully monitor and manage their condition. To prevent possible organ damage to the foetus, women with Type I Diabetes or Type II Diabetes should achieve target glucose levels before conception.

II. SUBSEQUENT GESTATIONAL DIABETES:

Once you've Gestational Diabetes in one pregnancy, you're more likely to have it again with the next pregnancy. [65]

DIAGNOSIS:

Diabetes Mellitus refers to a condition in which circulating blood glucose is chronically elevated. In Anaemia, there are many possible causes for a high blood glucose. Equally, there can be wide variety of possible consequences, such that a physician specializing in Diabetes must have some familiarity with almost every system in the body. Investigation of Diabetes itself can be divided into the study of its causes, natural history, epidemiology, genetic basis, pathophysiological mechanism and biochemical consequences, and each of its many complications requires investigating along similar lines. This entry provides a brief introduction to the way in which research findings have over the years been translated into the clinical investigation of Diabetes. [66]

- **Blood:**

1. Blood sugar estimation:
    a. Blood sugar estimation is mandatory for confirming the diagnosis of Diabetes.
    b. Both fasting and postprandial blood sugar levels are estimated.
2. Criteria for diagnosis of Diabetes Mellitus:
    a. Random blood sugar>200mg/dl on two occasions,
    b. Fasting Plasma Glucose>126mg/dl and sustained elevation of plasma glucose concentration>200mg/dl after an oral glucose load of 75gm at 2 hours.
3. Screening by Fasting Glucose test:

    Fasting blood glucose determination is a screening test for Type II Diabetes Mellitus. It is recommended for all above age of 45 yrs and must be tested every 3 yrs and relatively earlier in overweight persons. A fasting plasma glucose value above 126mg/dl is certainly indicative of Diabetes Mellitus.

INTERPRETATION (VENOUS PLASMA GLUCOSE):

Condition	Fasting	2 hrs after Glucose load
1.Fasting Hyperglycaemia	110-125 mg/dl	<140mg/dl
2.Impaired Glucose Tolerance	<126mg/dl	140-199mg/dl
3.Diabetes	>126mg/dl	>200mg/dl

Impaired glucose patients have increased risk of progression to frank Diabetes and Macrovascular atheromatous disease and kept under observation for repeating the test.

GLYCOSYLATED HAEMOGLOBIN (HbA1C):

Measurement of blood glucose level in Diabetics suffers from variation due to dietary intake of the previous day. Glycated Haemoglobin (HbA1C) provides an accurate and objective measure of glycaemic control over a period of weeks to months. In Diabetes the slow non-enzymatic covalent attachment of glucose to Haemoglobin (glycation) which takes place over 90-120 days that is lifespan of red blood cells. So it gives an estimate of Diabetic control for the preceding 3-4 months.There is an increase in the amount of the HbA_1 relative to Non-Glycated Adult Haemoglobin.

- Advantages of HbA1C:
  1. No dietary preparation or fasting is required
  2. Increased (HbA1C) value certainly means Diabetes Mellitus, but normal value does not rule out IGT
  3. It is not used to diagnose Diabetes but it gives idea about poor control and development of microvascular complications.
- **Normal value:**

  Below 7 %

  HbA1C diminished in
  1. Anaemia
  2. During pregnancy
  3. Uraemia
  4. Hemoglobinopathies
  5. Blood transfusions.

C-PEPTIDE ASSAY:

C-peptide is released into circulation during conversion of proinsulin to insulin which is more sensitive than insulin assay.

OTHER INVESTIGATIONS:

1. Lipid profile
2. Liver function test
3. Blood urea
4. Serum Creatinine

- **URINE:**

Glucose:

a. Testing the urine for glucose with dipsticks is a common screening procedure
b. for detecting Diabetes.
c. Performed on urine passed 1-2 hrs after meal to maximize sensitivity.
d. Glycosuria warrants further assessment by blood testing.

Common cause for Glycosuria :

Low renal threshold which also occur in pregnancy, starvation, raised intracranial tension (cerebral tumours, haemorrhage and head injury) and alimentary glycosuria.

Renal Glycosuria:

Normal renal threshold for glucose is below 180 mg/dl but glucose still appear in urine due to low renal threshold. It is benign condition unrelated to Diabetes Mellitus and runs in families, also in pregnancy.

Alimentary Glycosuria:

A rapid and transitory rise in blood glucose level after meal above the normal renal threshold is called lag storage curve or alimentary glycosuria and returns to normal after 2 hrs.

Ketones:

Test for Ketone bodies is for assessing severity of Diabetes and not diagnosis of disease. If Glycosuria and Ketonuria are present, diagnosis of diabetes is certain, Ketonuria also seen in fasting, strenuous exercise, diet rich in fat and low in carbohydrate.

Proteins:

Microalbuminuria or Proteinuria in absence of urinary tract infection is an indicator of development of Diabetic Nephropathy and other macrovascular complications. [67]

MANAGEMENT:

The management of Type I and II DiabetesMellitus (DM) requires addressing multiple goals, with the primary goal being glycemic control. Maintaining glycemic control in patients with Diabetes prevents many of the microvascular and macrovascular complications associated with Diabetes. This chapter presents a review of the prevalence, screening, diagnosis, and management of these complications.

- **MICROVASCULAR:**

Microvascular complications of Diabetes are those long-term complications that affect small blood vessels. These typically include Retinopathy, Nephropathy, and Neuropathy.

- Diabetic Retinopathy is divided into two main categories:
  Non-proliferative Retinopathy and Proliferative Retinopathy.
  - Non-proliferative Retinopathy is the development of Microaneurysms, venous loops, Retinal Haemorrhages, hard exudates, and soft exudates.
  - Proliferative Retinopathy is the presence of new blood vessels, with or without vitreous Haemorrhage. It is a progression of Non-Proliferative Retinopathy.
- Diabetic Nephropathy is defined as persistent Proteinuria, which is characterized by progressive decline in renal function resulting in end-stage renal disease.
- Diabetic Neuropathy is a heterogeneous condition associated with nerve pathology. The condition is classified according to the nerves affected and includes focal, diffuse, sensory, motor, and autonomic Neuropathy.

- **MACROVASCULAR:**

Macrovascular complications of Diabetes are primarily diseases of the coronary arteries, peripheral arteries, and cerebrovasculature. Early macrovascular disease is associated with atherosclerotic plaque in the vasculature supplying blood to the heart, brain, limbs, and other organs. Late stages of macrovascular disease involve complete obstruction of these vessels, which can increase the risks of Myocardial Infarction (MI), stroke, claudication, and gangrene. CardioVascular Disease (CVD) is the major cause of morbidity and mortality in patients with Diabetes.

The aim of treatment is to achieve normal blood glucose levels, to alleviate symptoms and to prevent complications.

The four pillars of Diabetic management are,

1. Diet
2. Exercise
3. Drugs –Oral hypoglycaemic agents and Insulin therapy by regular monitoring of glycaemic control.
4. Early detection and treatment of complications. [68]

1. DIET:

It is the cornerstone of management of Diabetes.The objective is to have good glycaemic control and to provide a nutritious and balanced diet. In Type II Diabetes the calories need to be restricted in order to avoid obesity.

Total Caloric Intake:

It depends on body weight, degree of physical activity and presence of Co - morbid illness.

Body Mass Index (BMI):

It determines the total caloric requirement

BMI= Weight (in kg) / Height in m^2.

BMI Normal Range**:**

22- 25

The calories are derived from three principal sources like carbohydrates, proteins and fats.

Carbohydrates:

The amount of carbohydrate recommended in the diet is upto 50-60%. Whole grains, ragi, wheat, millets, oats, brown rice which have low glycaemic index are recommended.

Proteins:

Recommended amount is 12-20% of total calorie intake. Dhals or grams with outer skin and sprouts, lean meat, fish, egg white and chicken are preferred.

Fat:

It should be 20-24% of total intake. Sunfloweroil, gingely oil, safflower oil, olive oil rich in Mono and Polyunsaturated fats are advised. Palm oil, coconut oil and vanaspathi should be avoided.

Salt:

Dietary salt should be less than 6g/day.

Milk and Milk Products:

Contribute to 40-45% of total fat content of vegetarian diet. Skimmed milk, unsweetened yogurt, curd, buttermilk are recommended.

Vegetables:

Fibre rich in greens, brinjal, cauliflower, gourds and salads are advised.

PHYSICAL ACTIVITY:

Exercise forms an important component along with drugs and diet management in Type II Diabetes Mellitus. Patients should be encouraged to take regular physical activity in form of walking, jogging, swimming, gardening and cycling for 30 minutes daily. This improves insulin sensitivity, prevents complications of Diabetes, and assist in maintaining lipid profile and blood pressure, improves muscle strength and beneficial for mental state of the individual. [69]

TRIAL DRUG

PREPARATION OF TRIAL DRUG

Drug Name - Pungampoo Chooranam

Ingredients - Pungam flowers

Cow's Ghee

Standard Operative Procedure:

The shade dried flowers of Pungam tree are roasted slowly by adding little bit of cow's ghee. Then it is powdered and sieved using cloth.

Dosage : 2 gm/ Bd

Adjuvant : Warm water

Duration : 48 Days

Text Reference - Boga Munivar Vaithiyam – 700

LITERATURE REVIEW OF PUNGAMPOO CHOORANAM

பாரப்பா மேகமிரு பதுவுந்தீரப்
பாடுகின்றேன் புங்கம் பூவென்ற மூலி
நேரப்பா ஒருபடிப்பூ வாரிவந்து
நேயமா யாய்ந்தெடுத்துப் பாண்டத்திட்டு
சேரப்பா வடுப்பேற்றி யெரித்துக் கிண்டு
சிறப்பாக வாவினெய்படி தானென்று
ஊரப்பா வறுக்குமப்போ கொஞ்சம் கொஞ்சம்
உண்ணஉண்ண பார்த்துமே புரட்டிவாங்கே

வாங்கியே யொருநேரம் வெருகடித்தூள்
மண்டலந்தான் கொண்டிடவே யந்நோயெல்லாம்
ஏங்கியே மேகவகை யெல்லா மோடும்
இலவுபட்ட தீப்போல வெரிந்துபோகும்

பாங்கியே பத்தியந்தான் பகரக்கேளு

பசிவான புளிப்புகையும் தள்ளவேண்டும்

ஈங்கியதோர் வாயுவென்ற பதார்த்தந்தள்ளி

இச்சாபத்தியமாக வுண்டு தேறே

– போக முனிவர் வைத்தியம் 700 [70]

Botanical name	-	*Pongamia pinnata*
Family	-	Fabaceae (Papilionaceae)
Part used	-	Flower
Taste (Suvai)	-	Kaippu
Nature (Thanmai)	-	Veppam
Classification (Pirivu)	-	Kaarppu
Action	-	Astringent, Alternative, Parasiticide

CHEMICAL CONSTITUENTS:

- Karanjin
- Pongapin
- 3 - Methoxypongapin
- Pongaglobrone
- Kanjone
- Pongol
- Gamatin
- Lanceolatin 3
- Pinnatin
- 5 – Methoxy – 3’, 4’ Methylenedioxyfurano
- Ionchocarpin
- Isolonchocarpin
- Karanjachromene
- Pongachromene
- Isopongachromene
- Pongaflavone

- Isopongaflavone
- Ovalichromene B
- Pongamol
- Ovalitenone
- Glabrin
- Kanugin
- Cemethoxykanugin
- Fisetintetramethylether
- Glabrachalcone
- Glabrachalcone – I
- Glabrachalcone – II
- Pongachalcone – I
- Pongachalcone – II
- Isopongaglabol
- B – Sitosterol
- Quercetin glycoside
- Kaempferol and its glycoside
- Pongamin
- Neoglabrin
- Glabrasaponin
- Sterolin
- Fatty acid of the seed oil has been reported.

The aqueous extract of *PongammiaPinnata* flowers shows Antidiabetic activity in streptozotocin induced diabetic rats.

COW'S GHEE:

Pasu Nei Gunam:

தாகமுழலைகட்கஞ்சர்த்தி பித்தம்வாயுப்பிர
மேகம் வயிற்றெரிவு விக்கலழல் – மாகாசங்
குன்மம் வறட்சி குடற்புரண்ட லஸ்திசுட்கஞ்
சொன்மூலம் போக்குநிரை துப்பு. [71]

Thanmai - Thatpam.

CHEMICAL CONSTITUENTS:

Saturated fatty acids

Triglycerides

Diglycerides

Monoglycerides

Phospholipids

Beta carotene 600 IU and vitamin E.

Action:

It controls thirst, vomiting, excessive pitha, burning sensation of the stomach, pitha hiccup, abdominal pain, dryness, prickly heat, cough, hyper motility of the gut, weakness of bones, haemorrhoids etc.

TRIAL DRUG – PUNGAMPOO CHOORANAM

DRIED PUNGAM FLOWERS

COW'S GHEE

PUNGAMPOO CHOORANAM

MATERIALS AND METHODS

MATERIALS AND METHODS

STUDY DESIGN:

The clinical trial on Madhumegam (Diabetes Mellitus – Type II) was decided to conduct as an open label study.

STUDY CENTER:

The entire study was conducted on patients at Out Patient Department of Govt Siddha Medical College, Chennai in the premises of Arignar Anna government hospital for Indian medicine and Homeopathy, Arumbakkam, Chennai-106, during the period of 2015-2017.

DATA COLLECTION:

Literary evidence from various,

- Siddha books
- Modern books
- Medical journals
- Internet

POPULATION:

The Population consists of Diabetes accompanied by Polyuria, Polyphagia, Polydipsia, generalised tiredness, Fatigue, Peripheral neuritis, Itching all over the body and satisfying the inclusion and exclusion criteria mentioned below.

SAMPLE SIZE:

40 patients.

INCLUSION CRITERIA:

- Newly identified Type II Diabetic cases only.
- Subject within 30-60 years.
- Blood Glucose (F) – 126mg/dl to 140 mg/dl
- Blood Glucose (pp) - 180mg/dl to 280 mg/dl
- HbA1C - 6.5% to 8%
- Polyuria

- Polyphagia
- Polydipsia
- Nocturia
- Fatigue

EXCLUSION CRITERIA (BASED ON CLINICAL HISTORY):

- H/O Insulin Dependent Diabetes Mellitus (IDDM).
- H/O Cardiovascular Disease.
- H/O Diabetic Nephropathy.
- H/O Diabetic Retinopathy.
- Pregnant women, lactating mothers, T.B affected individuals.

DURATION OF TREATMENT:

48 days.

Patients were followed under the guidance and supervision of the HOD, Professor, Reader, Lecturer and Assistant Lecturer of Maruthuvam, PG Department, GSMC, Chennai-106.

The patients were carefully studied for their history, clinical examinations, investigations and management.

EVALUATION OF CLINICAL PARAMETERS:

The history includes past, personal, family, occupation, dietary habits and associated history.

CLINICAL INVESTIGATION:

- Blood:

Blood sugar (Fasting, Post Prandial)

Glycaemic control: HbA1C

Blood urea

Serum creatinine

Serum cholesterol

- BMI (Body Mass Index)
- Urine:

  Urine Sugar (Fasting, Postprandial)

SIDDHA ASSESSMENT:

Envagaithervugal

Neerkuri

Neikkuri

A case sheet format was prepared based on the Siddha methodology like Envagaithervugal, Mukkutram, Nilam, Kaalam, Udalthathukkal including Neerkuri and Neikuri. Individual case sheet was maintained for each patient at Outpatient Department.

RESULTS
AND
OBSERVATION

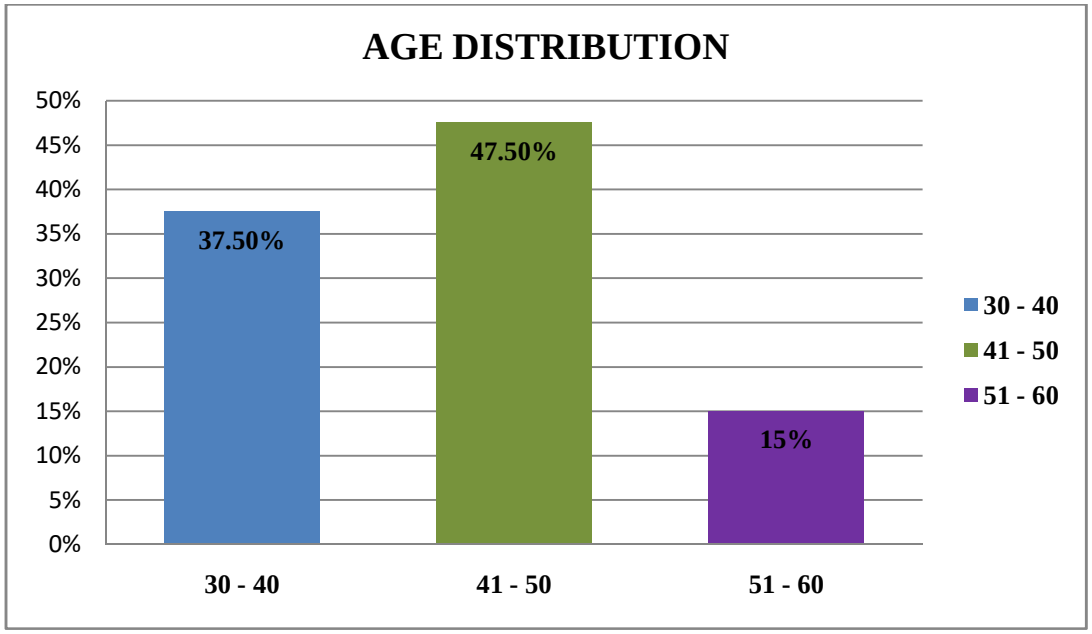
RESULTS AND OBSERVATION

The study on Madhumegam was carried out with 40 patients in the Out Patient Department PG Maruthuvam, Govt Siddha Medical College attached to Arignar Anna Govt hospital of Indian medicine, Chennai-106, during the period 2015-2017 were analysed. The observation were made and tabulated with following criteria.

1. Age Distribution
2. Sex Distribution
3. Occupational Status
4. Socio economic Status
5. Dietary Habits
6. Family History
7. Kaalam
8. Paruva Kaalam
9. Thinnai
10. Duration of illness
11. Mukkutram - Vali, Azhal, Iyam
12. Ezhu Udalthathukkal
13. Ennvagai Thervugal
14. Naadi
15. Neikurai
16. Clinical features
17. Clinical Prognosis
18. Urine sugar - Fasting, PostPrandial
19. Blood Sugar - Fasting, PostPrandial
20. HbA1C Level
21. Grading of Results.

1. AGE DISTRIBUTION

Age	No. Of Cases	Percentage
30 – 40	15	37.5%
41 – 50	19	47.5%
51 - 60	6	15%

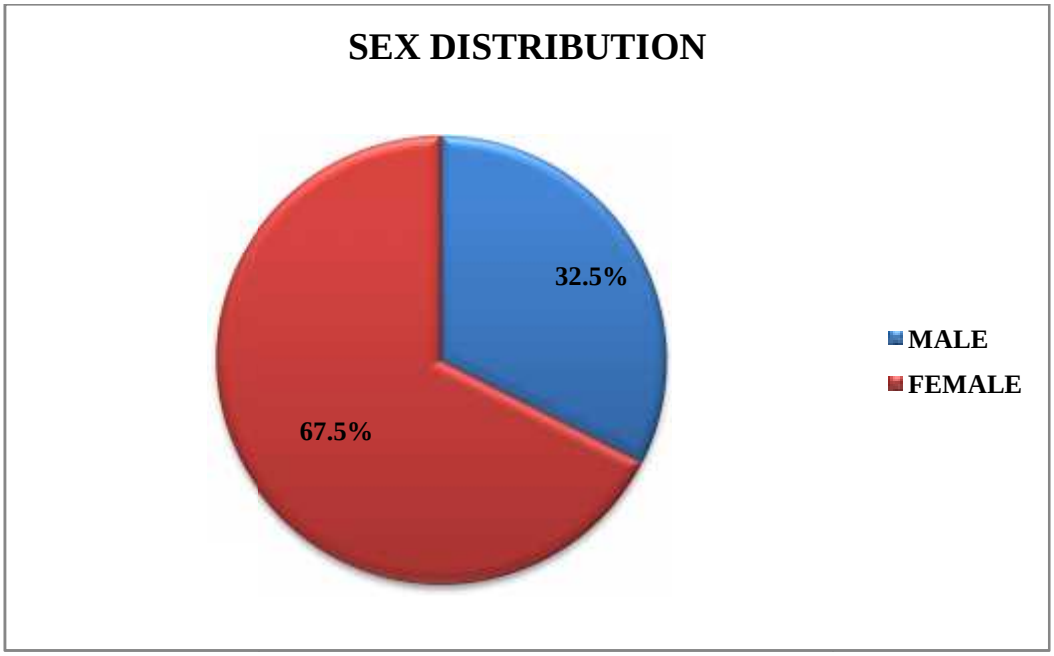


Inference:

From selected 40 cases, 15 patients (37.5%) were between 30 - 40 years, 19 patients (47.5%) were between 41 – 50 years and 6 patients (15%) were between 51 – 60 years old.

2. SEX DISTRIBUTION:

Sex	No. Of Cases	Percentage
Male	13	32.5%
Female	27	67.5%

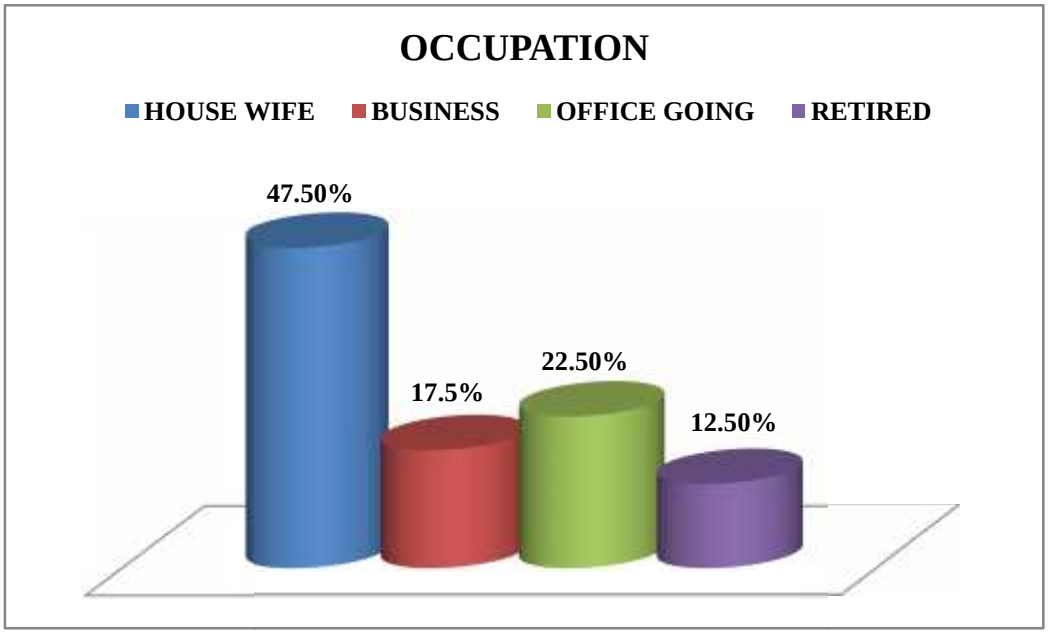


Inference:

Out of 40 patients, 13 cases (32.5%) were male and 27 cases (67.5%) were female.

3. OCCUPATIONAL STATUS:

Occupation	No. Of Cases	Percentage
House Wife	19	47.5%
Business	7	17.5%
Office Going	9	22.5%
Retired	5	12.5%

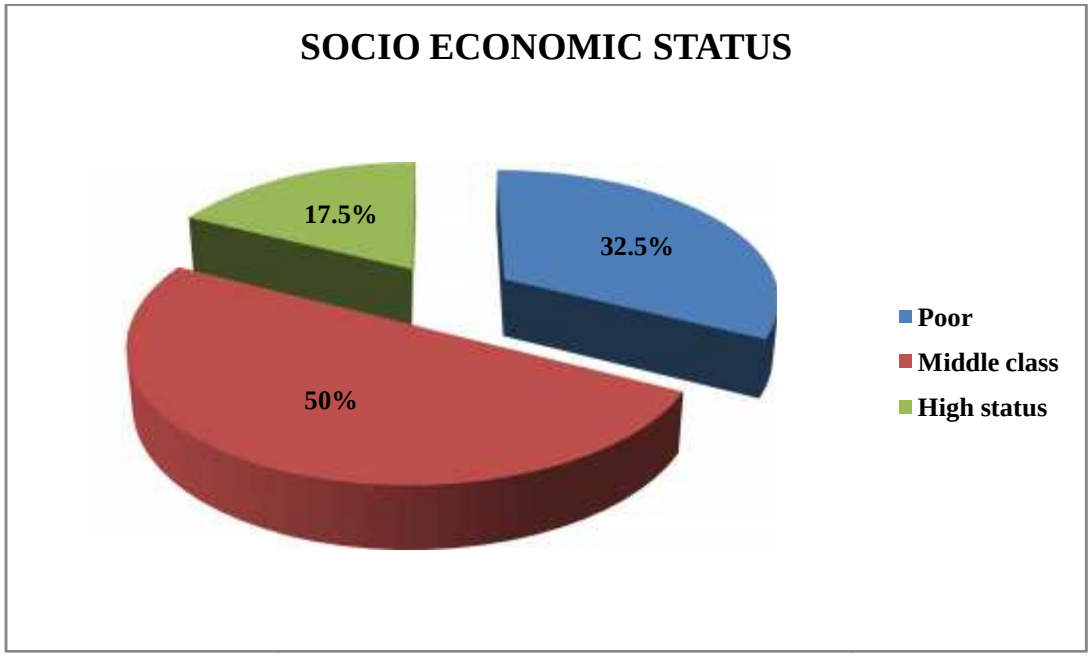


Inference:

From selected 40 cases, 19 patients (47.5%) were housewives, 7 patients (17.5%) are doing business, 9 patients (22.5%) are office goers and 5 (12.5%) are retired.

4. SOCIO ECONOMIC STATUS:

Socio Economic Status / Annum	No. Of Cases	Percentage
Poor (Upto Rs.2,00000)	13	32.5%
Middle Class (Rs.2,00000- 5,00000)	20	50%
High Class (Above Rs.5,00000)	7	17.5%

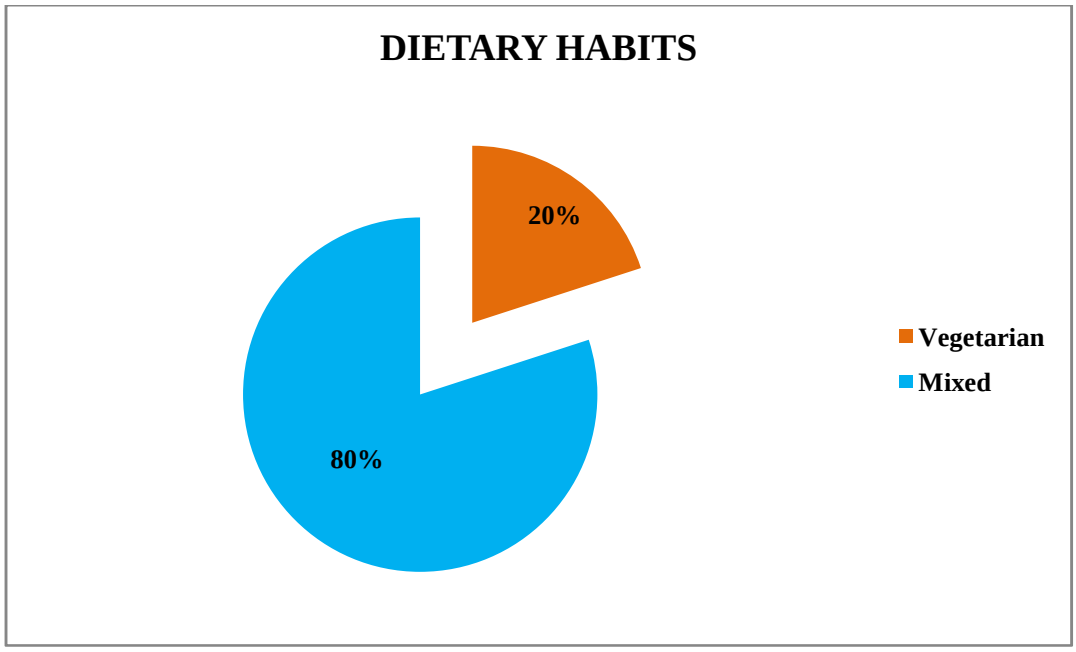


Inference:

Regarding Socio Economic Status 13 Patients (32.5%) comes under Poor category, 20 patients (50%) come under Middle class and 7 Patients (17.5) comes under High status.

5. DIETARY HABITS:

Dietary Habits	No. Of Cases	Percentage
Vegetarian	8	20%
Mixed	32	80%

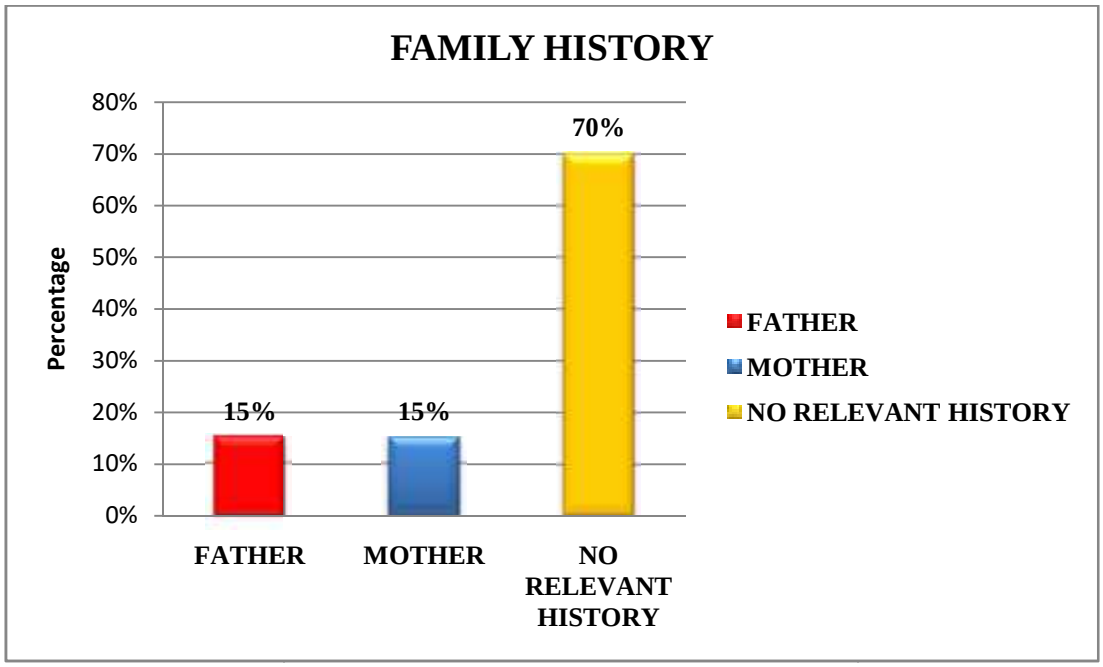


Inference:

Regarding Diet, out of 40 patients, 8 patients (20%) takes Vegetarian diet and 32 patients (80%) takes mixed diet.

6. FAMILY HISTORY:

Family History	No. Of Cases	Percentage
Father	6	15%
Mother	6	15%
No Relevant History	28	70%

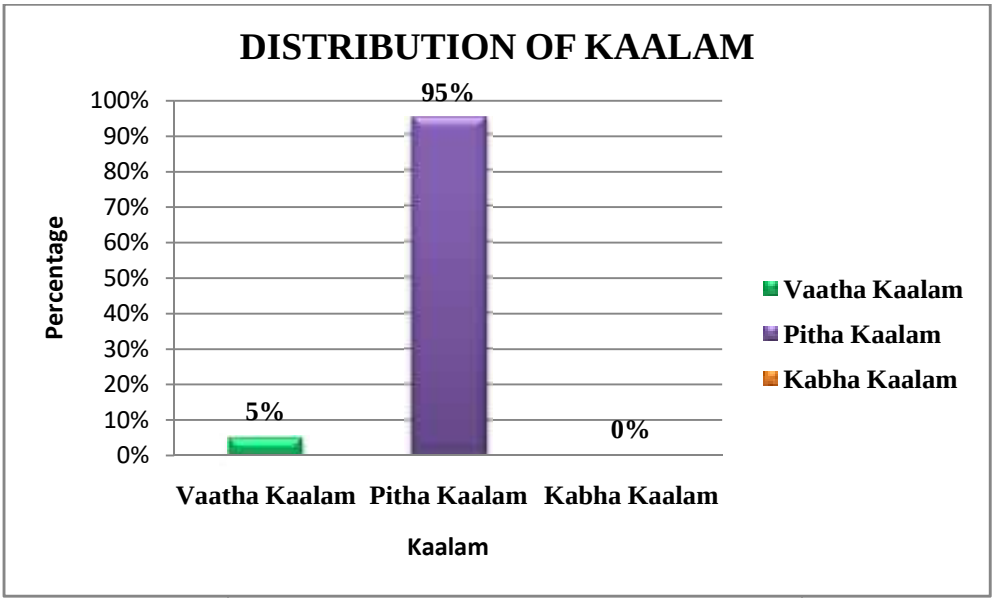


Inference:

Regarding family history 6 patients (15%) fathers with diabetic, 6 patients (15%) mothers are diabetic and 28 patients (70%) have no relevant family history.

7. DISTRIBUTION OF KAALAM:

Kaalam	No. Of Cases	Percentage
Vaatha Kaalam (0- 33 years)	2	5%
Pitha Kaalam (34-66 years)	38	95%
Kabha Kaalam (67-100 years)	Nil	0%

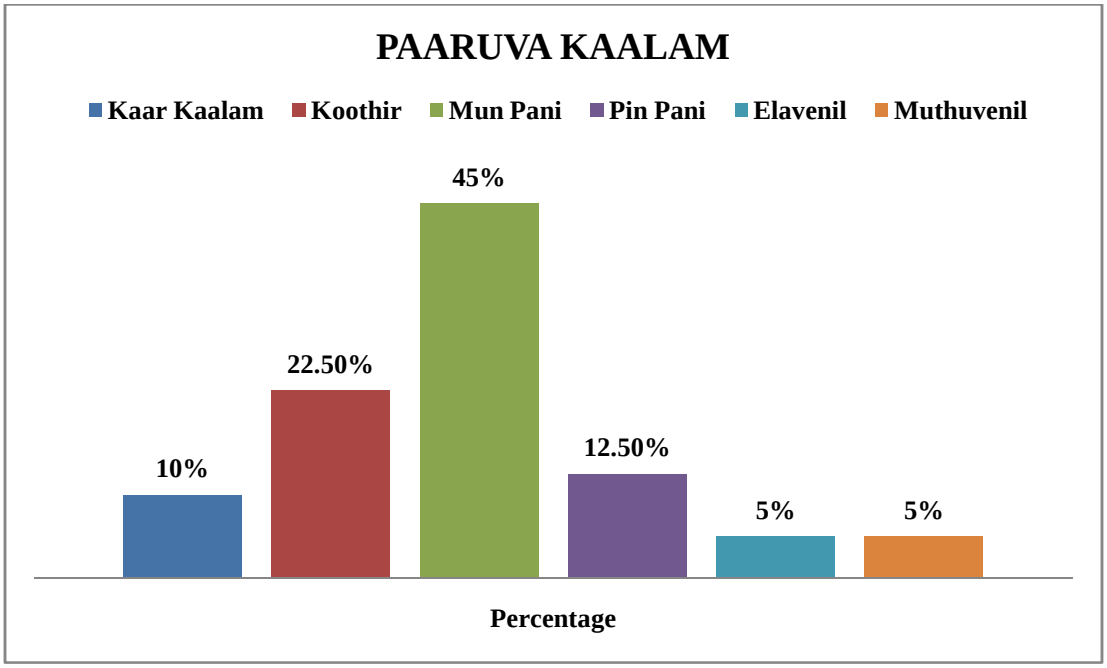


Inference:

Out of 40 patients, 2 patients (5%) comes under Vaatha Kaalam, 38 patients (95%) comes under Pitha Kaalam and no patients comes under Kabha Kaalam.

8. PARUVA KAALAM:

Paruva Kaalam (Seasons)	Month	No. Of Cases	Percentage
Kaar Kaalam	Aavani, Purattasi (Mid Aug- Mid Oct)	4	10%
Koothir Kaalam	Iyppasi, Karthigai (Mid Oct – Mid Dec)	9	22.5%
Mun Pani Kaalam	Margazhi, Thai (Mid Dec – Mid Feb)	18	45%
Pin Pani Kaalam	Maasi, Panguni (Mid Feb – Mid Apr)	5	12.5%
Elavenil Kaalam	Chithirai, Vaigasi (Mid Apr – Mid June)	2	5%
Muthu Venil Kaalam	Aani, Aadi (Mid June – Mid Aug)	2	5%

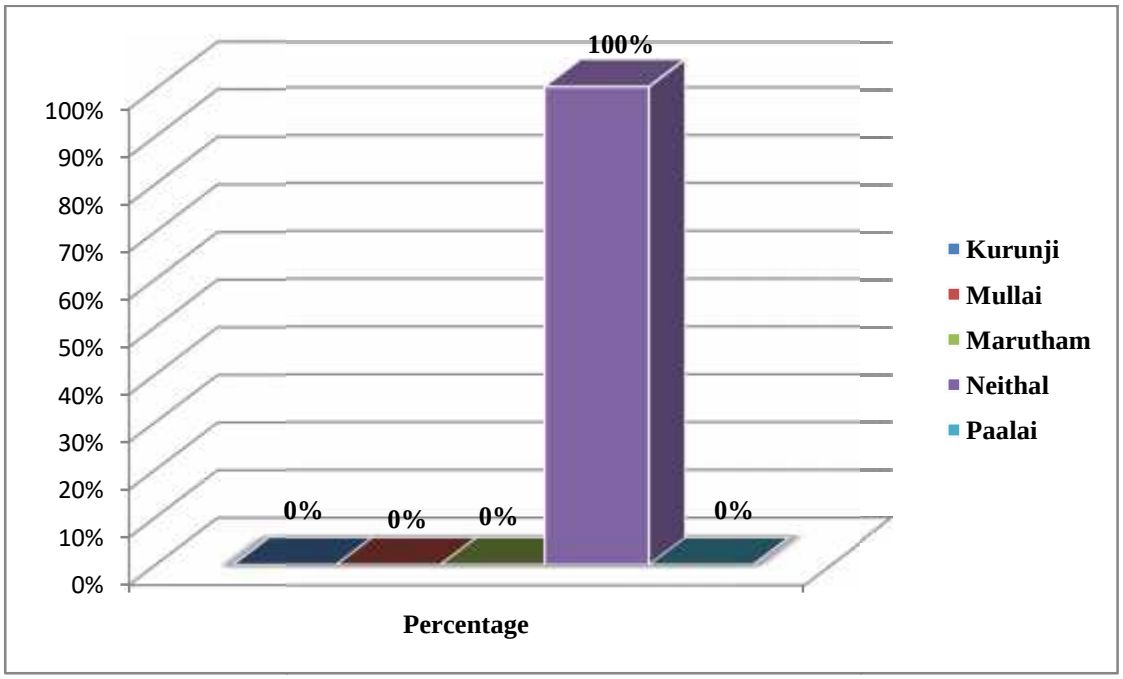


Inference:

From selected 40 patients, 4 patients (10%) comes under Kaar Kaalam, 9 patients (22.5%) comes under Koothir Kaalam, 18 patients (45%) comes under Mun Pani Kaalam, 5 patients (12.5%) comes under Pin Pani Kaalam, 2 patients (5%) comes under Elavenil Kaalam and 2 patients (5%) comes under Muthuvenil Kaalam.

9. DISTRIBUTION OF THINAI:

Thinai	No. Of Cases	Percentage
Kurunji	0	0%
Mullai	0	0%
Marutham	0	0%
Neithal	40	100%
Paalai	0	0%

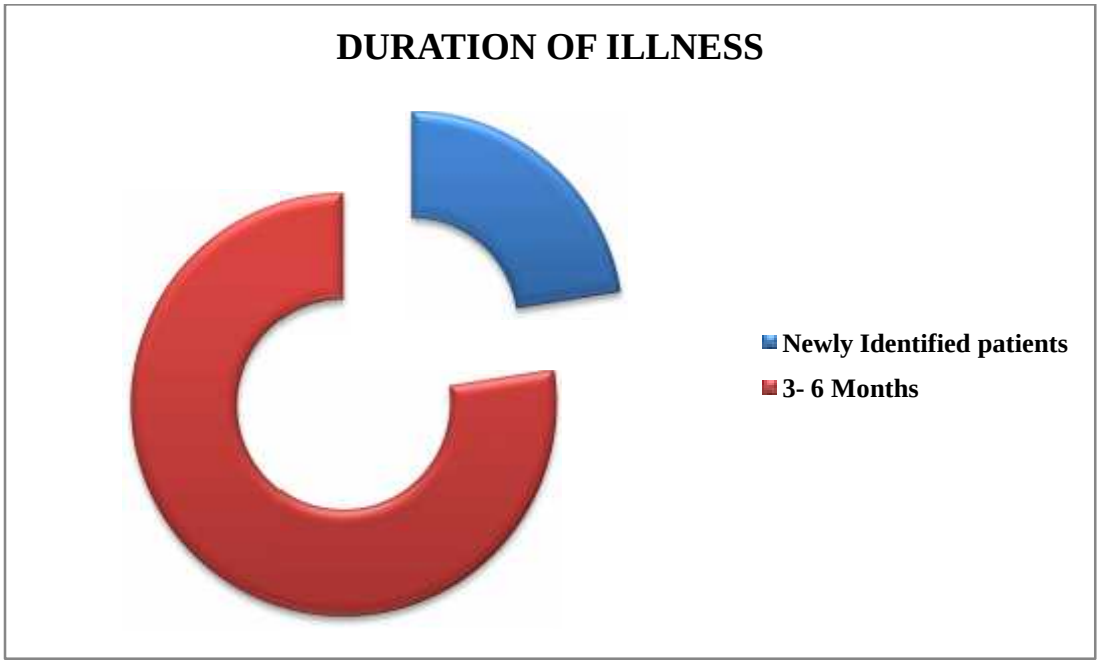


Inference:

All the 40 patients belongs to Neithal Nilam.

10. DURATION OF ILLNESS:

Duration Of Illness	No. Of Cases	Percentage
Newly Identified patients	9	22.5%
3- 6 Months	31	77.5%



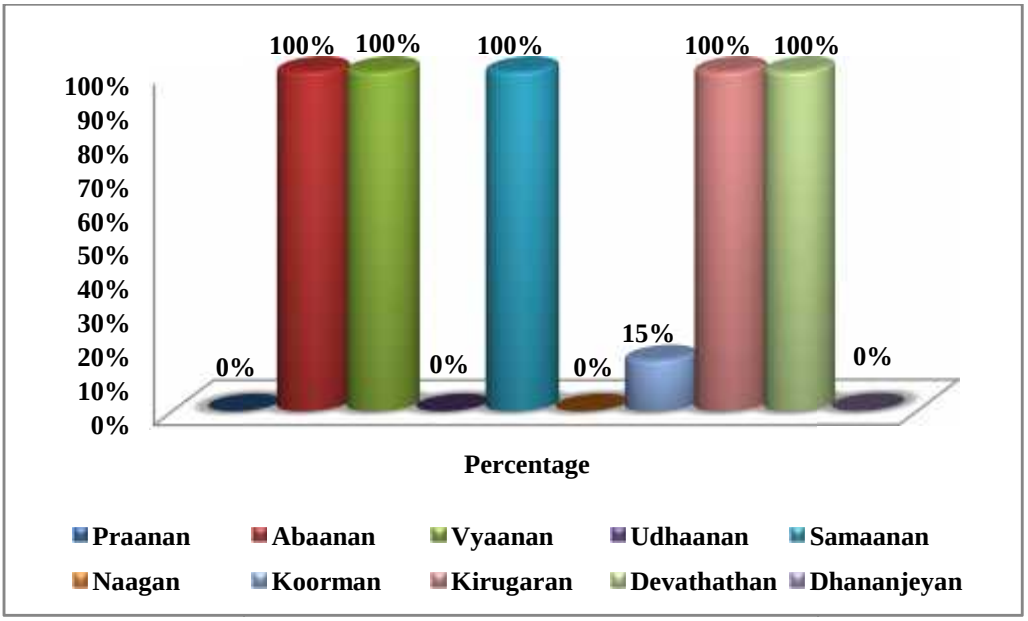
Inference:

Out of 40 patients, 9 patients (22.5%) belong to newly identified category and 31 patients (77.5%) belongs to 3 – 6 months category.

11. REFERENCE TO MUKKUTTRAM:

a. Affected Vali:

Classification Of Vali	No. Of Cases	Percentage
Praanan	0	0%
Abaanan	40	100%
Vyaanan	40	100%
Udhaanan	0	0%
Samaanan	40	100%
Naagan	0	0%
Koorman	6	15%
Kirugaran	40	100%
Devathathan	40	100%
Dhananjeyan	NIL	0%

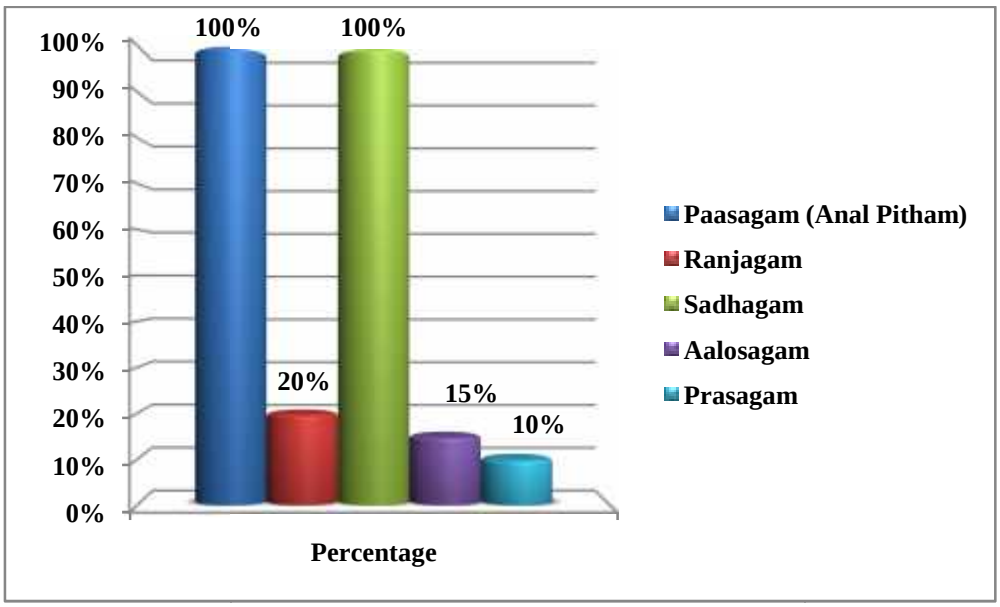


Inference:

From the selected 40 patients, all the 40 patients were affected with Abaanan, Vyaanan, Samaanan, Kirugaran and Devathathan, 6 patients (15%) was affected with Koorman and none affected with Praanan, Udhaanan, Naagan and Dhananjeyan.

b. Affected Azhal:

Classification Of Azhal	No. Of Cases	Percentage
Paasagam (Anal Pitham)	40	100%
Ranjagam	8	20%
Sadhagam	40	100%
Aalosagam	6	15%
Prasagam	4	10%

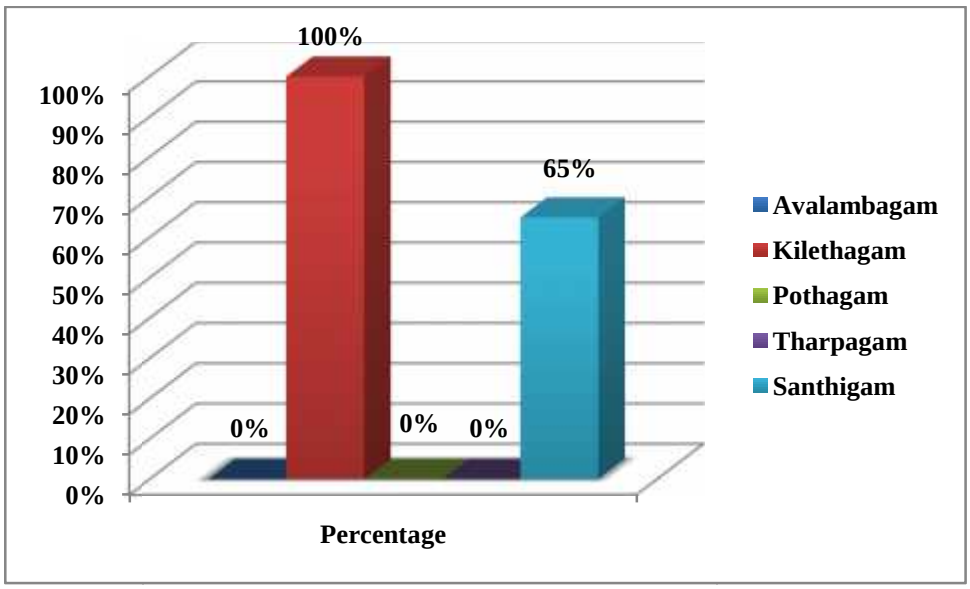


Inference:

Out of 40 patients, all the 40 patients were affected with Paasagam and Saadhagam, 8 patients (20%) was affected with Ranjagam, 6 patients (15%) was affected in Aalosagam and 4 patients (10%) was affected with Prasagam.

c. Affected Iyyam:

Classification Of Iyyam	No. of Cases	Percentage
Avalambagam	0	0%
Kilethagam	40	100%
Pothagam	0	0%
Tharpagam	0	0%
Santhigam	26	65%

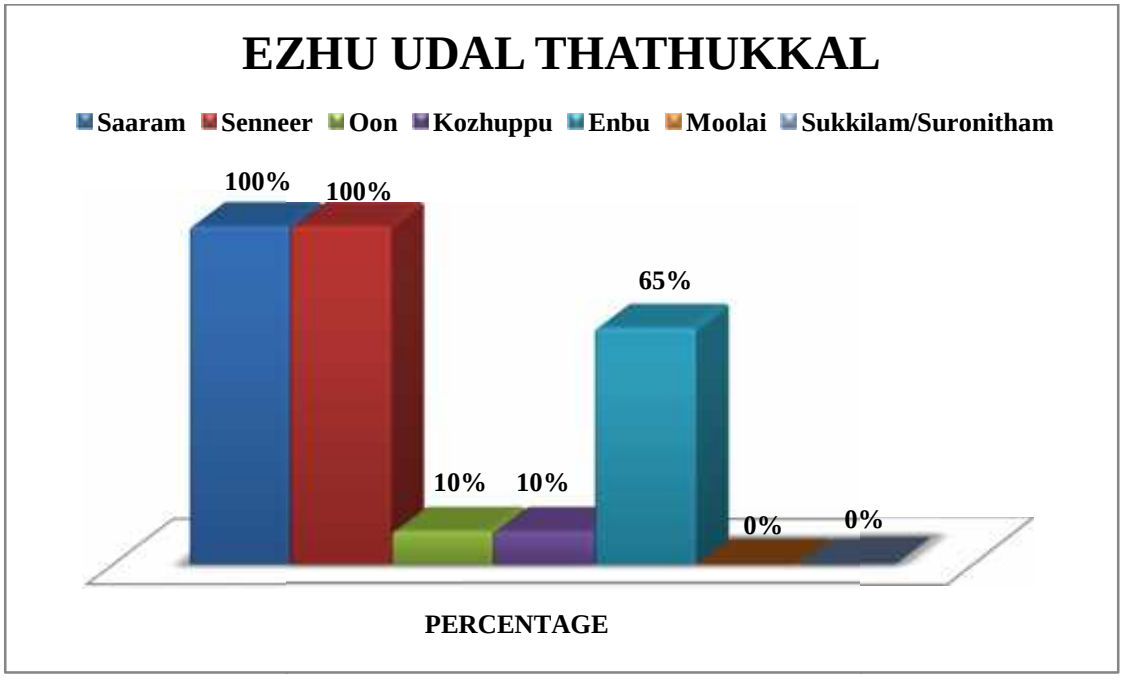


Inference:

Out of 40 patients, all patients were affected with Kilethagam, Santhigam was affected in 26 patients (65%) and none affected with Avalambagam, Pothagam and Tharpagam.

12. EZHU UDAL THATHUKKAL:

Ezhu Udal Thathukkal	No. Of Cases	Percentage
Saaram	40	100%
Senneer	40	100%
Oon	4	10%
Kozhuppu	4	10%
Enbu	26	65%
Moolai	0	0%
Sukkilam/Suronitham	0	0%

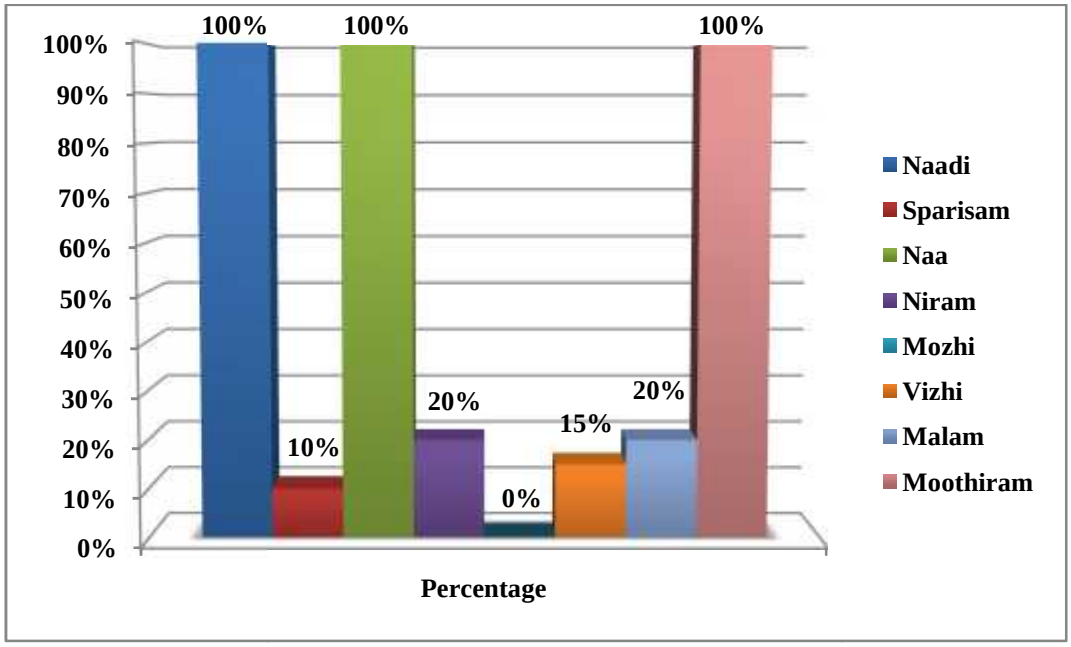


Inference:

From the above chart, we observe that Saaram and Senneer were affected in all the patients (100%), Oon & Kozhuppu was affected to the extent of 10% and Enbu affected in 26 patients (65%). None affected with Moolai and Sukkilam.

13. ENN VAGAI THERVUGAL:

Enn Vagai Thervugal	No. Of Cases	Percentage
Naadi	40	100%
Sparisam	4	10%
Naa	40	100%
Niram	8	20%
Mozhi	0	0%
Vizhi	6	15%
Malam	8	20%
Moothiram	40	100%

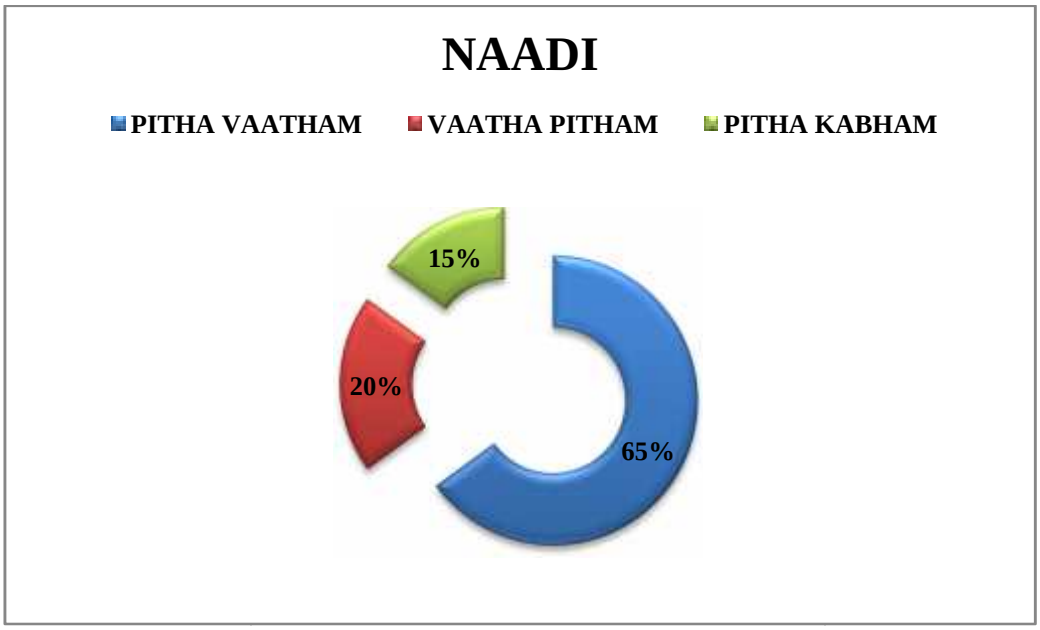


Inference:

Regarding Enn Vagai Thervu, none was affected with Mozhi. 8 patients (20%) was Malam and Niram affected, Vizhi was affected in 6 patients (15%), 4 patients (10%) was sparisam affected, Naadi, Naa and Moothiram was affected for all the 40 patients.

14. NAADI:

Naadi	No. Of Cases	Percentage
Pitha Vaatham	26	65%
Vaatha Pitham	8	20%
Pitha Kabham	6	15%

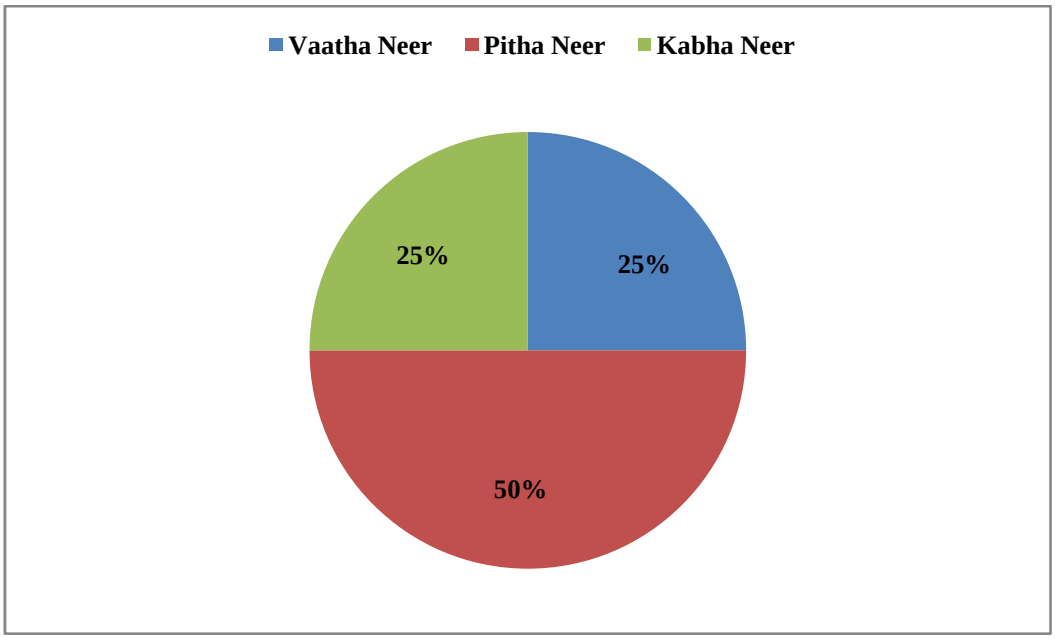


Inference:

Out of 40 patients, 26 patients (65%) had Pitha Vaatha naadi, 8 patients (20%) had Vaatha Pitha naadi and 6 patients (15%) had Pitha Kabha naadi.

15. NEI KURI REFERNCE:

Nei Kuri	Character Of Urine	No. Of Cases	Percentage
Vaatha Neer	Spreads like snake	10	25%
Pitha Neer	Spreads like ring	20	50%
Kabha Neer	Float like pearl	10	25%



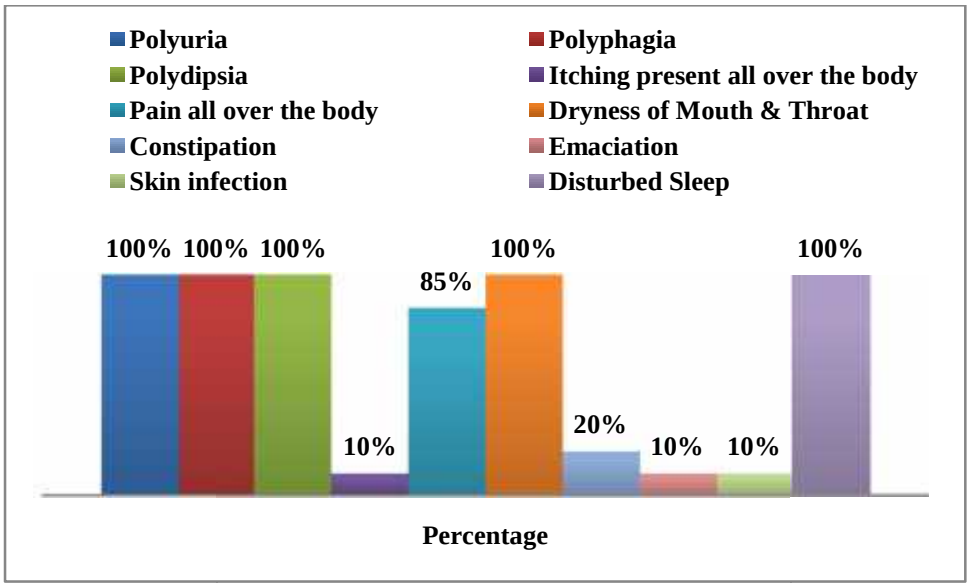
Inference:

Out of 40 patients, 10 patients (25%) had Vaatha Neer, 20 patients (50%) had Pitha Neer and 10 patients (25%) had Kabha Neer.

16. CLINICAL FEATURES:

Signs & Symptoms	No. Of Cases	Percentage
Polyuria	40	100%
Polyphagia	40	100%
Polydipsia	40	100%
Itching present all over the body	4	10%
Pain all over the body	34	85%
Dryness of Mouth & Throat	40	100%
Constipation	8	20%
Emaciation	4	10%
Skin infection	4	10%
Disturbed Sleep	40	100%

SIGNS & SYMPTOMS



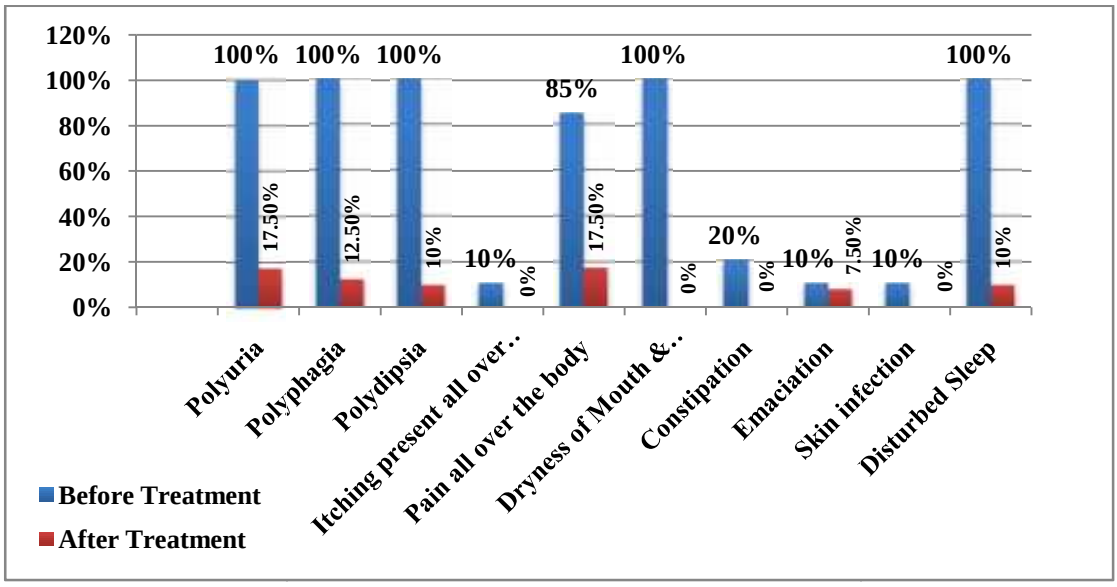
Inference:

In respect of the patients with Mathumegam, the clinical symptoms of Polyuria, Polyphagia, Polydipsia, Dryness of Mouth & Throat and Disturbed sleep were present in all cases (100%). Itching all over the body, skin infection & Emaciation in 4 patients (10%), Pain all over the body in 34 cases (85%), Constipation in 8 patients (20%).

17. CLINICAL PROGNOSIS:

Signs & Symptoms	Before Treatment		After Treatment	
	No. Of Cases	Percentage	No. Of Cases	Percentage
Polyuria	40	100%	7	17.5%
Polyphagia	40	100%	5	12.5%
Polydipsia	40	100%	4	10%
Itching present all over the body	4	10%	0	0%
Pain all over the body	34	85%	7	17.5%
Dryness of Mouth & Throat	40	100%	0	0%
Constipation	8	20%	0	0%
Emaciation	4	10%	3	7.5%
Skin infection	4	10%	0	0%
Disturbed Sleep	40	100%	4	10%

SIGNS & SYMPTOMS

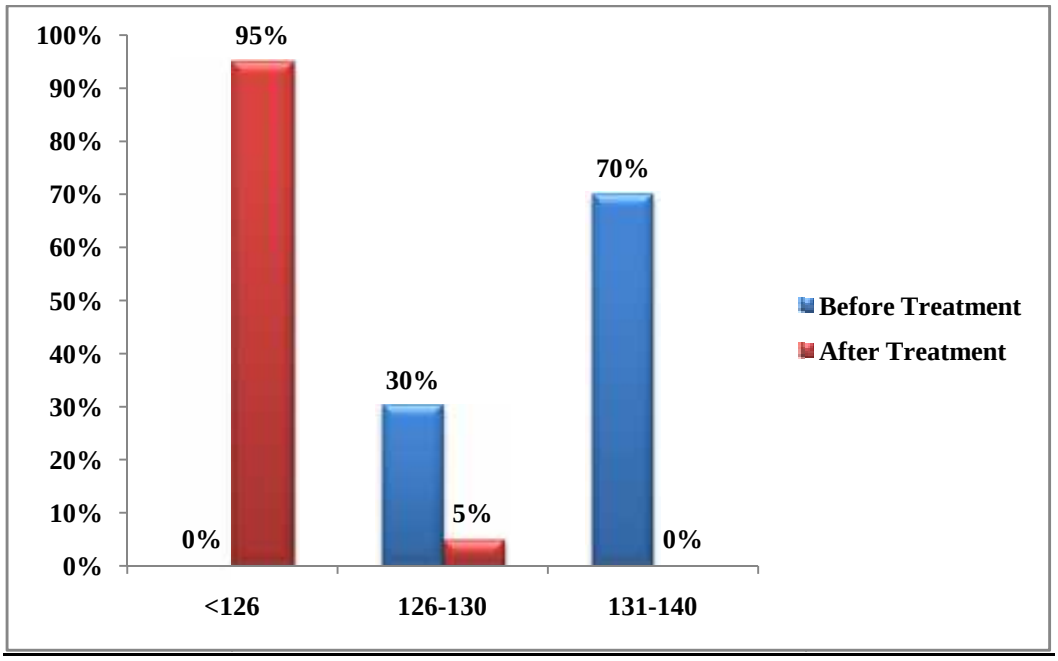


Inference:

The clinical signs & symptoms were improved after treatment, showing only 17.5% of people have Polyuria, 12.5% have Polyphagia, 10% of people have Polydipsia, 17.5% had pain all over the body, 10% have skin infection and 10% had disturbed sleep 7.5% had emaciation. The symptoms of Itching all over the body, dryness in Mouth & Throat, constipation, skin infection were completely relieved.

18.A) BLOOD SUGAR (FASTING):

Blood Sugar mg/dl	Before Treatment (No. Of Cases)	Percentage	After Treatment (No. Of Cases)	Percentage
<126	0	0%	38	95%
126-130	12	30%	2	5%
131-140	28	70%	0	0%

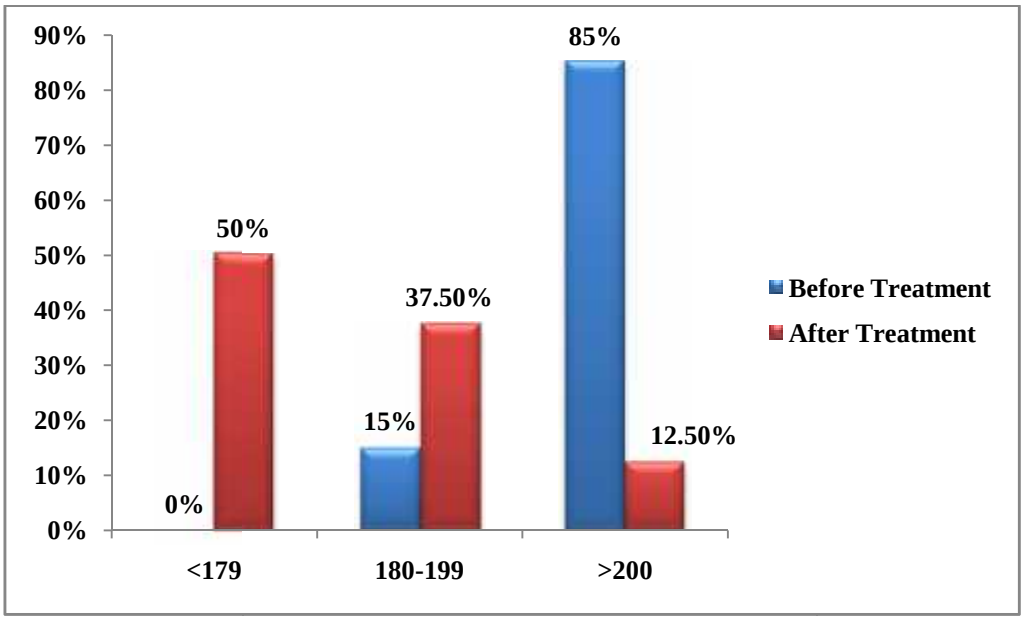


Inference:

Fasting blood sugar has controlled by 95% of cases.

18.(B) BLOOD SUGAR (POST PRANDIAL):

Blood Sugar mg/dl	Before Treatment (No. Of Cases)	Percentage	After Treatment (No. Of Cases)	Percentage
<179	0	0%	20	50%
180-199	6	15%	15	37.5%
>200	34	85%	5	12.5%

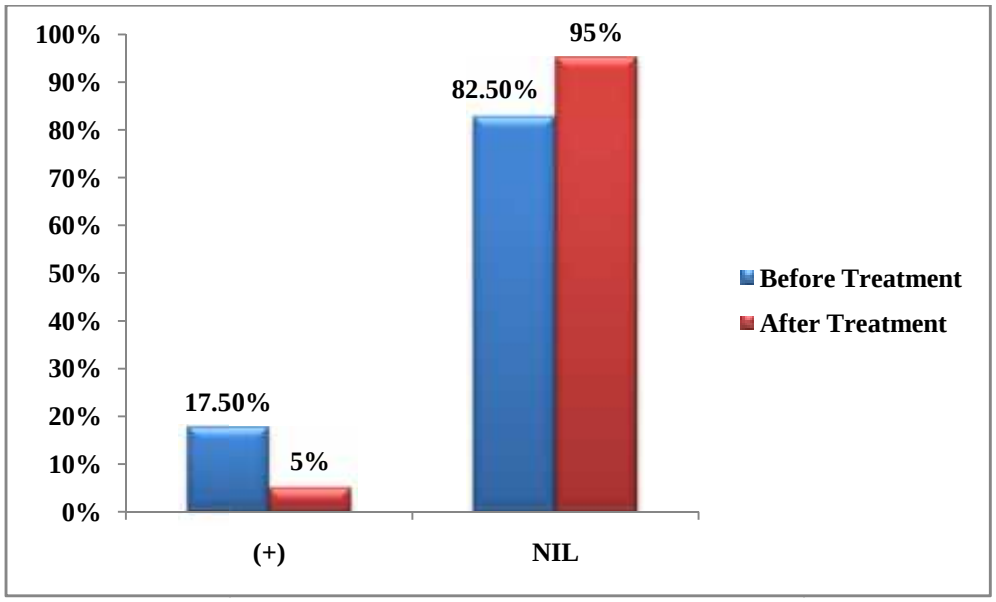


Inference:

The Blood Sugar Post Prandial has controlled by 87.5% of cases.

19.(A)URINE SUGAR (FASTING)

Urine Sugar	Before Treatment (No. Of Cases)	Percentage	After Treatment (No. Of Cases)	Percentage
(+)	7	17.5%	2	5%
NIL	33	82.5%	38	95%

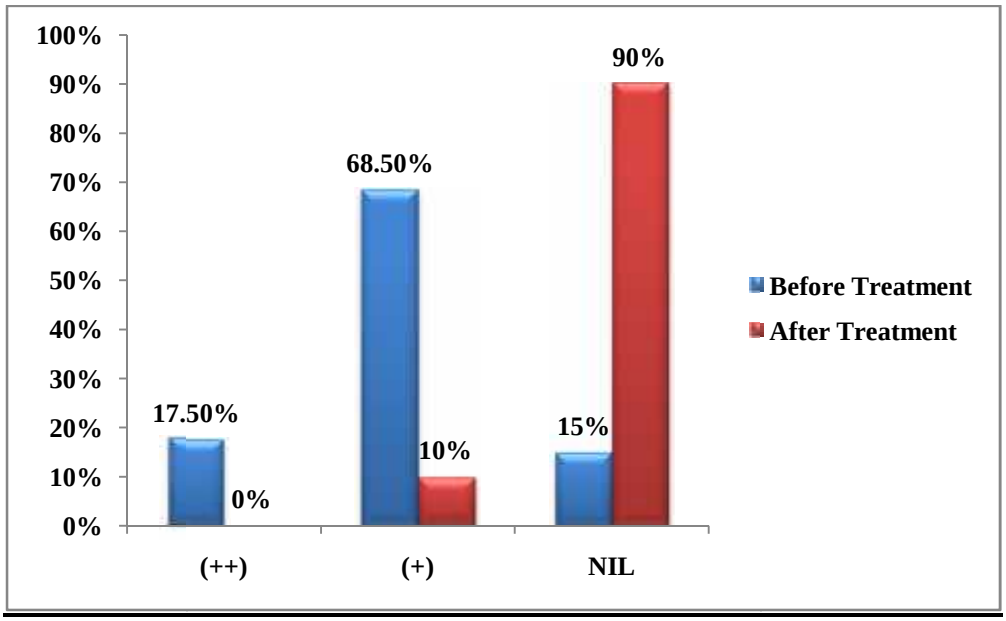


Inference:

From the above chart it may be observed that urine sugar position on fasting after treatment had improved drastically. And it was nil in 95% of cases after treatment.

19. (B) URINE SUGAR (POST PRANDIAL):

Urine Sugar	Before Treatment (No. Of Cases)	Percentage	After Treatment (No. Of Cases)	Percentage
(++)	7	17.5%	0	0%
(+)	27	68.5%	4	10%
NIL	6	15%	36	90%

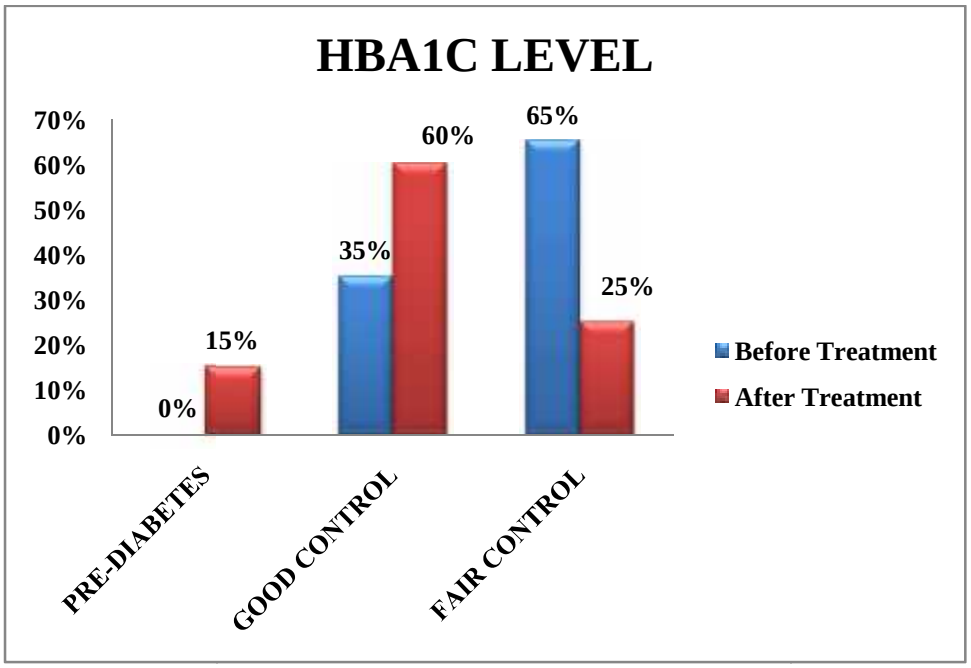


Inference:

It may be noted that post prandial urine sugar position after treatment had improved drastically. It was nil in 90% of cases after treatment.

20. HBA1C LEVEL:

HBA1C	Before Treatment (No. Of Cases)	Percentage	After Treatment (No. Of Cases)	Percentage
Pre-Diabetes	0	0%	06	15%
Good Control	14	35%	24	60%
Fair Control	26	65%	10	25%

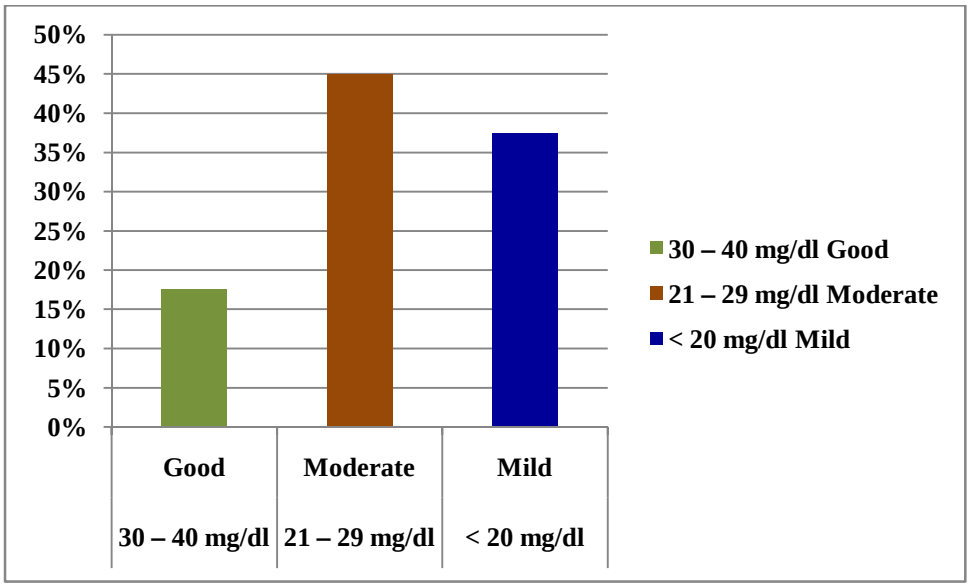


Inference:

HBA1C level has good control in 75% of cases and fair control in 25% of cases after treatment.

21. (A). BASED ON REDUCTION IN BLOOD SUGAR FASTING:

Blood Sugar Level (F)	Prognosis	No. Of Cases	Percentage
30 – 40 mg/dl	Good	7	17.5%
21 – 29 mg/dl	Moderate	18	45%
< 20 mg/dl	Mild	15	37.5%

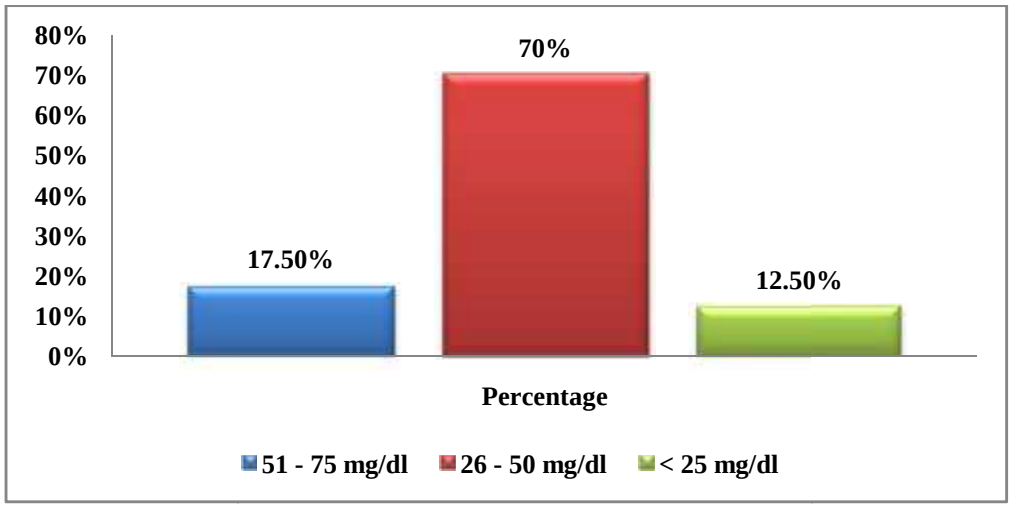


Inference:

Out of 40 patients, 7 patients (17.5%) shows good result, 18 patients (45%) shows moderate results and 15 patients (37.5%) shows mild results.

21 (B). BASED ON REDUCTION IN BLOOD SUGAR POST PRANDIAL:

Blood Sugar Level (PP)	Prognosis	No. Of Cases	Percentage
51 - 75 mg/dl	Good	7	17.5%
26 - 50 mg/dl	Moderate	28	70%
< 25 mg/dl	Mild	5	12.5%



Inference:

Out of 40 patients, 7 patients (17.5%) shows good result, 28 patients (70%) shows moderate results and 5 patients (12.5%) shows mild results.

BLOOD SUGAR LEVEL

S.NO	OUT PATIENT NO.	AGE/ SEX	BLOOD SUGAR LEVEL			
			BEFORE TREATMENT		AFTER TREATMENT	
			F (mg/dl)	PP (mg/dl)	F (mg/dl)	PP (mg/dl)
1	5994	40/F	132	231	110	193
2	4957	45/M	138	233	120	191
3	4948	40/M	136	221	122	188
4	5783	44/F	131	202	119	168
5	491	31/M	136	189	94	152
6	2194	48/M	139	242	124	218
7	2382	40/M	132	214	121	180
8	2452	40/F	129	230	97	188
9	5107	45/M	133	214	109	181
10	5402	38/F	130	219	106	186
11	6905	37/F	127	183	103	161
12	7529	50/F	140	263	132	217
13	7567	40/F	129	220	98	159
14	8031	37/F	126	227	98	161
15	8704	50/M	139	247	105	196
16	9008	35/F	133	188	111	143
17	9939	48/F	137	213	113	177
18	300	36/F	127	185	95	157
19	815	58/F	129	218	119	169
20	995	47/F	138	220	114	173
21	1379	47/F	133	203	120	171
22	1691	45/F	131	226	109	180
23	1692	48/F	128	214	94	179
24	1968	54/M	135	245	108	208
25	2008	55/F	138	253	122	188
26	7720	60/M	140	258	130	203
27	3100	50/F	136	206	121	174
28	3138	30/F	131	186	105	166
29	3436	47/M	128	219	108	179
30	4577	46/M	136	226	114	187
31	4612	50/F	128	221	106	176
32	4660	40/F	133	228	117	179
33	4664	38/M	130	219	102	186
34	4732	46/F	128	228	97	176
35	5280	55/F	138	257	119	221
36	5610	37/F	131	184	98	168
37	7617	45/M	136	231	112	173
38	8218	53/F	133	222	123	182
39	8252	44/F	131	229	113	197
40	8657	43/F	134	237	119	181

BMI CHART OF THE PATIENTS

			BEFORE TREATMENT				AFTER TREATMENT			
S.No	**O.P.No.**	**Age/ Sex**	**Wt (kg)**	**Ht (CM)**	**BMI**	**H/L/O**	**Wt (kg)**	**Ht (CM)**	**BMI**	**H/L/O**
1	5994	40/F	62	162	23	H	60	162	23	H
2	4957	45/M	58	160	23	H	57	162	22	H
3	4948	40/M	60	163	23	H	57	163	22	H
4	5783	44/F	70	165	26	O	69	165	25	O
5	491	31/M	59	158	23	H	56	158	22	H
6	2194	48/M	59	163	22	H	57	163	22	H
7	2382	40/M	68	173	22	H	65	173	22	H
8	2452	40/F	62	160	24	H	60	162	23	H
9	5107	45/M	65	170	22	H	63	170	22	H
10	5402	38/F	63	163	24	H	60	163	23	H
11	6905	37/F	50	164	18	L	55	164	20	H
12	7529	50/F	80	175	26	O	77	175	25	O
13	7567	40/F	70	163	26	O	68	163	26	O
14	8031	37/F	70	189	24	H	74	189	21	H
15	8704	50/M	65	163	24	H	62	163	23	H
16	9008	35/F	60	162	22	H	58	162	22	H
17	9939	48/F	70	170	24	H	67	170	23	H
18	300	36/F	75	170	26	O	73	170	25	O
19	815	58/F	56	157	22	H	54	157	22	H
20	995	47/F	61	165	22	H	59	165	22	H
21	1379	47/F	63	163	24	H	62	163	23	H
22	1691	45/F	55	155	24	H	52	155	22	H

			BEFORE TREATMENT				AFTER TREATMENT			
S.No	**O.P.No.**	**Age/ Sex**	**Wt (kg)**	**Ht (CM)**	**BMI**	**H/L/O**	**Wt (kg)**	**Ht (CM)**	**BMI**	**H/L/O**
23	1692	48/F	70	170	24	H	68	170	23	H
24	1968	54/M	50	163	18	L	54	163	20	H
25	2008	55/F	56	157	22	H	54	157	22	H
26	7720	60/M	61	165	22	H	59	165	22	H
27	3100	50/F	58	163	22	H	56	163	21	H
28	3138	30/F	75	170	27	O	73	170	25	O
29	3436	47/M	50	163	18	L	55	163	21	H
30	4577	46/M	61	165	21	H	59	165	22	H
31	4612	50/F	59	163	22	H	57	163	21	H
32	4660	40/F	55	155	22	H	53	155	22	H
33	4664	38/M	72	170	24	H	70	170	24	H
34	4732	46/F	50	163	18	L	55	163	21	H
35	5280	55/F	61	168	21	H	65	168	23	H
36	5610	37/F	50	163	18	L	53	163	20	H
37	7617	45/M	70	165	26	O	68	165	25	O
38	8218	53/F	62	165	24	H	60	165	22	H
39	8252	44/F	61	165	21	H	58	165	21	H
40	8657	43/F	59	163	22	H	56	163	21	H

H – Healthy L – Lean O - Overweight

BIO CHEMICAL ANALYSIS OF THE PATIENTS

S.No	O.P. NO.	Age/ Sex	Blood Sugar Level (mg/dl)				Urine Sugar Level				HBA1C	
			BT		AT		BT		AT			
			F	PP	F	PP	F	PP	F	PP	BT	AT
1	5994	40/F	132	231	110	193	NIL	(+)	NIL	NIL	7.1	6.4
2	4957	45/M	138	233	120	191	(+)	(+)	NIL	NIL	7.3	6.2
3	4948	40/M	136	221	122	188	NIL	(+)	NIL	NIL	7.0	6.3
4	5783	44/F	131	202	119	168	NIL	(+)	NIL	NIL	6.9	6.4
5	491	31/M	136	189	94	152	NIL	NIL	NIL	NIL	6.6	5.2
6	2194	48/M	139	242	124	218	(+)	(++)	NIL	(+)	8.0	7.9
7	2382	40/M	132	214	121	180	NIL	(+)	NIL	NIL	7.3	6.5
8	2452	40/F	129	230	97	188	NIL	(+)	NIL	NIL	7.2	6.4
9	5107	45/M	133	214	109	181	NIL	(+)	NIL	NIL	7.3	6.3
10	5402	38/F	130	219	106	186	NIL	(+)	NIL	NIL	6.9	6.1
11	6905	37/F	127	183	103	161	NIL	NIL	NIL	NIL	6.7	5.8
12	7529	50/F	140	263	132	217	(+)	(++)	(+)	(+)	8.0	7.8
13	7567	40/F	129	220	98	159	NIL	(+)	NIL	NIL	7.8	6.5
14	8031	37/F	126	227	98	161	NIL	(+)	NIL	NIL	7.1	6.4
15	8704	50/M	139	247	105	196	(+)	(++)	NIL	NIL	7.8	6.3
16	9008	35/F	133	188	111	143	NIL	NIL	NIL	NIL	6.7	5.7
17	9939	48/F	137	213	113	177	NIL	(+)	NIL	NIL	7.0	6.5
18	300	36/F	127	185	95	157	NIL	NIL	NIL	NIL	6.8	5.7
19	815	58/F	129	218	119	169	NIL	(+)	NIL	NIL	7.3	6.5
20	995	47/F	138	220	114	173	NIL	(+)	NIL	NIL	7.4	6.8
21	1379	47/F	133	203	120	171	NIL	(+)	NIL	NIL	6.8	6.4

S.NO	O.P.NO	Age/ Sex	Blood Sugar Level (mg/dl)				Urine Sugar Level				HBA1C	
			BT		AT		BT		AT			
			F	PP	F	PP	F	PP	F	PP	BT	AT
22	1691	45/F	131	226	109	180	NIL	(+)	NIL	NIL	7.0	6.7
23	1692	48/F	128	214	94	179	NIL	(+)	NIL	NIL	8.0	7.9
24	1968	54/M	135	245	108	208	NIL	(++)	NIL	(+)	7.8	7.5
25	2008	55/F	138	253	122	188	NIL	(++)	NIL	NIL	7.2	7.0
26	7720	60/M	140	258	130	203	(+)	(++)	(+)	(+)	8.0	7.6
27	3100	50/F	136	206	121	174	(+)	(+)	NIL	NIL	6.8	6.3
28	3138	30/F	131	186	105	166	NIL	NIL	NIL	NIL	6.7	5.8
29	3436	47/M	128	219	108	179	NIL	(+)	NIL	NIL	7.3	6.5
30	4577	46/M	136	226	114	187	NIL	(+)	NIL	NIL	7.4	6.8
31	4612	50/F	128	221	106	176	NIL	(+)	NIL	NIL	7.6	7.3
32	4660	40/F	133	228	117	179	NIL	(+)	NIL	NIL	7.5	7.1
33	4664	38/M	130	219	102	186	NIL	(+)	NIL	NIL	6.8	6.1
34	4732	46/F	128	228	97	176	NIL	(+)	NIL	NIL	7.9	7.6
35	5280	55/F	138	257	119	221	(+)	(++)	NIL	(+)	8.0	7.8
36	5610	37/F	131	184	98	168	NIL	NIL	NIL	NIL	7.1	5.9
37	7617	45/M	136	231	112	173	NIL	(+)	NIL	NIL	7.6	6.9
38	8218	53/F	133	222	123	182	NIL	(+)	NIL	NIL	8.0	7.3
39	8252	44/F	131	229	113	197	NIL	(+)	NIL	NIL	7.0	6.4
40	8657	43/F	134	237	119	181	NIL	(+)	NIL	NIL	6.9	6.6

BT – Before Treatment, AT- After Treatment, N – Nil, F- Fasting, PP – Post Prandial

LABORATORY INVESTIGATION REPORT OF THE PATIENTS

S.No	OP .No	Age/Sex	Blood Urea (mg/dl)		Serum Creatinine (mg/dl)		Total Cholesterol (mg/dl)	
			BT	AT	BT	AT	BT	AT
1	5994	40/F	26	23	0.6	0.7	176	172
2	4957	45/M	27	29	0.7	0.6	179	180
3	4948	40/M	32	30	0.86	0.82	174	180
4	5783	44/F	21	22	0.74	0.72	185	176
5	491	31/M	38	29	0.92	0.75	188	186
6	2194	48/M	42	40	0.9	0.86	179	181
7	2382	40/M	28	32	0.86	0.85	191	187
8	2452	40/F	39	21	0.86	0.61	179	178
9	5107	45/M	32	32	0.67	0.92	176	186
10	5402	38/F	31	29	0.72	0.69	167	189
11	6905	37/F	19	24	0.65	0.52	176	142
12	7529	50/F	24	24	0.72	0.69	161	184
13	7567	40/F	26	21	0.59	0.71	167	176
14	8031	37/F	26	24	0.74	0.81	166	149
15	8704	50/M	44	24	0.86	0.86	181	190
16	9008	35/F	36	23	0.86	0.67	187	166
17	9939	48/F	47	40	0.68	0.75	179	173
18	300	36/F	21	18	0.84	0.56	179	105
19	815	58/F	38	32	0.74	0.86	166	178
20	995	47/F	32	31	0.76	0.67	179	173
21	1379	47/F	13	23	0.78	0.65	180	178
22	1691	45/F	21	27	0.86	0.76	182	165
23	1692	48/F	35	32	0.68	0.85	176	175
24	1968	54/M	41	38	0.89	0.86	173	182
25	2008	55/F	26	29	0.68	0.81	186	176
26	7720	60/M	41	24	0.86	0.91	179	178
27	3100	50/F	40	20	0.91	0.62	186	151
28	3138	30/F	20	27	0.62	0.80	165	181

S.No	OP .No	Age/Sex	Blood Urea (mg/dl)		Serum Creatinine (mg/dl)		Total Cholesterol (mg/dl)	
			BT	AT	BT	AT	BT	AT
29	3436	47/M	40	29	0.94	0.85	192	176
30	4577	46/M	23	29	0.89	0.80	190	167
31	4612	50/F	26	22	0.78	0.60	161	159
32	4660	40/F	26	28	0.82	0.70	172	160
33	4664	38/M	32	23	0.74	0.75	181	182
34	4732	46/F	31	21	0.82	0.60	188	180
35	5280	55/F	40	29	0.91	0.86	176	179
36	5610	37/F	32	18	0.86	0.75	169	181
37	7617	45/M	42	35	0.94	0.94	180	182
38	8218	53/F	40	28	0.86	0.75	179	186
39	8252	44/F	21	26	0.90	0.76	189	174
40	8657	43/F	28	23	0.74	0.73	160	159

BT – Before Treatment

AT – After Treatment

DISCUSSION

DISCUSSION

Diabetes Mellitus, a group of metabolic disorder in which a person has high blood sugar level, either because the pancreas does not produce enough Insulin, or because cells do not respond to the Insulin that is produced. This high blood sugar produces the classical symptoms of Polyuria (frequent urination), Polydipsia (increased thirst) and Polyphagia (increased hunger).

Madhumegam is a clinical entity described by Yugi Munivar in 'Yugi Vaithya Chinthamani 800' can be compared with Diabetes Mellitus-Type II. The classical symptoms are Polyuria, Polyphagia, Polydipsia, itching all over the body and pain all over the body.

Various Siddha literatures has been studied and discussed for choosing the trial drug for treating Madhumegam and finally choosen "Pungampoo chooranam", which was mentioned in 'Boga Munivar Vaithyam 700'.

Authentication is a critical step for successful and reliable clinical applications and for further experimental studies on Siddha drugs.

DRUG AUTHENTICATION

Authentication of given specimen is the basic starting point in developing a botanical product.

A sample of specimen is collected from farm near Vandavasi and its organoleptic characters, Microscopic and Macroscopic examination was conducted and authenticated by botanist from G.S.M.C. Chennai.

PHYSICOCHEMICAL ANALYSIS:

Physicochemical parameters includes

Loss on drying at 105^0c	-	9.24%
Total ash	-	8.01%
Water soluble ash	-	4.75%
Acid insoluble ash	-	0.69%
Water soluble extractive	-	26.46%
Alcohol soluble extractive	-	29.35%
n - hexane soluble extractive	-	19.26%
pH value (10%)	-	7.5

These values of the given sample were compared with the standard values of Indian pharmacopoeia.

TOXICITY STUDY:

Toxicity studies in the animal models are done to determine and establish the dose level recommended for the treatment of disease as drug. Both acute and sub-acute toxicity studies are given special emphasis.

The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

IAEC Reference No**:** SU/CLATR/IAEC/IV/023/2016.

- **Acute Toxicity Study**

Acute toxicity study of the study drug Pungampoo Chooranam was carried out as per OECD guideline (Organization for Economic Co-operation and Development) Guideline-423. As a result of acute toxicity study no toxicity and mortality was found and subsequent study has been conducted.

- **Sub-Acute Toxicity Study**

Sub-acute toxicity of the study drug Pungampoo Chooranam was carried out as per OECD guideline-407. As a result of Sub-acute toxicity study no toxicity and mortality was found.

Hematological analysis:

When compared to the control group, treatment groups has no significant difference and hence treatment groups has no toxicity and haematological differences.

Biochemical analysis:

No significant difference between control and treatment groups and found no impacts in serological functions in treatment groups.

Histopathological evaluation:

Histopathology of vital organs like Liver, Kidney, Spleen and Lungs were carried out. This evaluation shows no toxic effect in the trial drug.

Statistical analysis:

The statistical analysis was carried by one way ANOVA (GRAPH PAD PRISM 5 computer program). Results were expressed as mean ± standard error. A statistical comparison was carried out using the Dunnet's test for the control and treatment group.

Based on the analysis the results show nullified values between the control group and treated groups.

PHARMACOLOGICAL EVALUATION:

The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

IAEC: SU/CLATR/IEAC/VII/051/2016.

Pharmacological studies of the trial drug Pungampoo Chooranam showed ANTI DIABETIC ACTIVITY on tested animals.

BIO CHEMICAL ANALYSIS:

Biochemical assays are needed to evaluate disease models and to drive biomarker analysis in translational medicine and clinical research.

Based on the analysis Pungampoo Chooranam exhibits the properties of reducing sugar, alkaloids, zinc and potassium.

IEC:

IEC has approved my trial drug with the allowed sample size of 40 patients with combined gender.

IEC No: GSMC-CH-ME-4/2015/009

CTRI:

The global mandate is to register all clinical trials prospectively, i.e. before the enrolment of the first patient. I have successfully registered my trial drug by submitting the details and scientific data's to Clinical Trial Registry.

CTRI NO: CTRI/2017/02/007956

CLINICAL STUDY:

Clinical studies were conducted followed by CTRI registration with the sample size of 40 patients.

In my study, 40 patients with Madhumegam were selected in the Department of Maruthuvam, Government Siddha Medical College, attached to Arignar Anna Govt Hospital for Indian Medicine, Arumbakkam, Chennai - 106.

All necessary investigations were carried out to all patients and trial medicine was given. The results of before and after treatment of all the patients were analysed and discussed below.

- **Age distribution:**
  - From selected 40 cases, 15 patients (37.5%) were between 30 - 40 years, 19 patients (47.5%) were between 41 – 50 years and 6 patients (15%) were between 51 – 60 years old.
  - Usually the non-insulin diabetes mellitus occurs only in the age group above 45 years – International Diabetic Monitor.
- **Sex distribution:**
  - Out of 40 patients, 13 cases (32.5%) were male and 27 cases (67.5%) were female. Recent studies show that more women are prone to diabetes than men.
- **Occupational status:**
  - From selected 40 cases, 19 patients (47.5%) were housewives, 7 patients (17.5%) are doing business, 9 patients (22.5%) are office goers and 5 (12.5%) are retired.
  - Incidence of Madhumegam is more in housewives. Nowadays due to modernisation and invention of electrical and electronic kitchen equipments, the women lack physical exercise and results in more prone to Madhumegam.
- **Socio-economic status**
  - Regarding Socio Economic Status 13 Patients (32.5%) comes under Poor category, 20 patients (50%) come under Middle class and 7 Patients (17.5) comes under High status.
  - People belonging to lower group are more prone to Madhumegam. Recent research indicates that the poor are more prone to diabetes.

Research was being conducted to analyze whether rapid changes in their lifestyle or the stress of poverty triggers diabetes.

- **Dietary Habits:**
  - Regarding Diet, out of 40 patients, 8 patients (20%) takes vegetarian diet and 32 patients (80%) takes mixed diet.
  - Madhumegam is more incidences on non-vegetarians. Further it could also be noted that people who are used fast and fried food are more prone to diabetes as they have more calories of fat.
- **Family history:**
  - Regarding family history 6 patients (15%) fathers with diabetic, 6 patients (15%) mothers are diabetic and 28 patients (70%) have no relevant family history. Genetics plays an important role in Madhumegam.
- **Paruvakaalam:**
  - From selected 40 patients, 4 patients (10%) comes under Kaar Kaalam, 9 patients (22.5%) comes under Koothir Kaalam, 18 patients (45%) comes under Mun Pani Kaalam, 5 patients (12.5%) comes under Pin Pani Kaalam, 2 patients comes under Elavenil Kaalam and 2 patients (5%) comes under Muthuvenil Kaalam.
  - The seasonal variation has no impact on Madhumegam.
- **Thinai:**
  - From the selected 40 patients, all (100%) comes under Neithal nilam .
- **Body built:**
  - Regarding body built, 29 patients (72.5%) were having normal weight, 6 patients (15%) were overweight and 5 patients (12.5%) were lean.
- **Duration of illness:**
    - Out of 40 patients, 9 patients (22.5%) belong to newly identified category and 31 patients (77.5%) belong to 3 – 6 months category.

MUKKUTRAM CLASSIFICATION:

❖ **In Vatham:**

- Abanan affected in all patients (100%) causing Polyuria, Nocturia .
- Viyanan affected in all patients (100%) with Pain all over the body.
- Samanan and Kirukaran affected in all patients (100%) causing Polyphagia.
- Devethathan affected in all patients (100%) causing disturbed sleep, fatigue.
- Koorman affected in 6 patients (15%) causing dimness of vision.

❖ **In Pitham:**

- Paasagam affected in all patients (100%) causing polyphagia.
- Saadhagam affected in all patients (100%) causing lassitude.
- Ranjagam and Prasagam affected in (20%), (10%) patients causing pallor and dry skin respectively.

❖ **In Kabham:**

- Kilethagam affected in all patients (100%) results in Polyphagia.
- Santhigam affected in 26 patients (65%) causing joint pain.

EZHU UDAL THATHUKKAL:

- Saaram affected in all patients (100%) results in tiredness, general debility.
- Senneer affected in all cases (100%) causing pallor, dryness.
- Oon and Kozhuppu affected in 4 patients (10%) each causing emaciation
- Enbu affected in 26 patients (65%) causing joint pain.

ENVAGAI THERVUGAL:

- Naa, Naadi and Moothiram affected in all 40 patients (100%).
- Malam affected in 8 patients (20%) results in constipation.
- Sparisam affected in 4 patients (10%) causing dry skin.
- Niram affected in 8 patients (20%) results in pallor of the skin.

NAADI:

- 26 patients (65%) had PithaVatha Naadi,
- 8 patients (20%) had Vatha Pitha Naadi and
- 6 patients (15%) had Pitha Kaba Naadi.

NEIKURI:

- 10 patients (25%) had Vathaneer,
- 20 patients (50%) had Pithaneer and
- 10 patients (25%) had Kabhaneer.

SIGNS AND SYMPTOMS:

- Polyuria, Polyphagia, Polydipsia, dryness of the mouth and throat and disturbed sleep present in all cases i.e 100%.
- Pain all over the body in 34 patients (85%),
- Itching in 10 patients (25%),
- Constipation in 8 patients (20%),
- Emaciation in 4 patients (10%) and
- Skin infection in10 cases (25%).

CLINICAL PROGNOSIS:

The clinical signs and symptoms were improved after treatment showing only 17.5% had polyuria, 12.5% had polyphagia, 10% of the people had polydipsia, 17.5% have pain all over the body, and 10% had disturbed sleep. Itching all over the body, skin infection, dryness of mouth and constipation were completely relieved.

LABORATORY ASSESSMENT:

- Blood sugar Fasting:
  - From the selected 40 patients, before treatment 28 patients are seen in the range of 131-140mg/dl and after treatment 38 patients are seen <126mg/dl.
- Blood sugar Post Prandial:
  - From the selected 40 cases before treatment 34 patients seen > 200 mg /dl and after treatment 20 patients post prandial sugar level were < 179 mg/ dl.
- Urine sugar Fasting:
  - From the selected 40 patients, before treatment 7 patients shows (+) and 33 patients shows Nil. After treatment 2 patients are reduced to (+) and 38 patients shows Nil Urine Sugar.
- Urine sugar Post Prandial:
  - From the selected 40 patients, before treatment 27 patients show (+) and 7 patients shows (++). After treatment 36 patients postprandial urine sugar shows Nil.
- HbA_1C:
  - From the selected 40 cases all patients are seen in the range of 6.5- 8 %. After treatment 30 patients had good control range of 6- 6.5 %.

SUVAI MUKKUTRAM THEORY:

Madhumegam is primarily due to derangement of Pitha kuttram. The trial medicine Pungampoo Chooranam predominant with Thuvarppu suvai, it neutralises the deranged pitham by Ethirurai Maruthuvam.

BIO STATISTICAL ANALYSIS:

The p value is highly significant ($p<0.001$). So, there is significant reducing of fasting, postprandial blood sugar level (mg) and HbA1C level among the patients for the treatment of Madhumegam. Hence, it is concluded that treatment was effective and significant.

GRADING OF RESULTS:

Out of 40 patients, 75% of cases showed Good result, 15% of the cases showed Moderate result and 10 % showed minimal significance.

SUMMARY

SUMMARY

The clinical study on Madhumegam was carried out in the Post Graduate Department of Maruthuvam, Govt Siddha Medical College, Arignar Anna Hospital, Chennai-106, during the period of 2015-2017.

A total of 40 patients were treated in the Out Patient Department (OPD). The clinical and pathological assessment was carried out on the basis of both Siddha and Modern aspects.

All the 40 patients were treated with Pungampoo Chooranam, 2g BD with warm water for 48 days. The responses were assessed once in 7days for all the patients.

- The peak incidence of Madhumegam was in the age group of 41-50 yrs (47.5%) in both sexes.
- The prevalence was higher among the poor (32.5%).
- The disease is more common in housewives (47.5%).so high incidence occurs in women.
- Regarding diet, the disease is seen among mixed dietary habits of about 80%.
- Regarding family history, 70% had no relavent history.
- Most of the patients were affected in Pitha kaalam (95%).
- In Vatham- Abaanan, Viyanan, Samanan, Kirukaran and Devathathan were affected in all the cases 100%, Koorman was affected in 6 cases (15%).
- In Pitham-Analagam and Sathagam 100%, Ranjagam 20%, Alosagam 15% and Prasagam 10% were affected.
- In Kabam- Kilethagam 100% and Santhigam 65% were affected.
- Among the Ezhu Udalthathukkal, Saaram, Seneer were 100% affected, Enbu (65%), Oon (10%), Kozhuppu (10%) were affected.
- Regarding Envagaithervugal - Naa, Naadi and Moothiram were 100% affected Niram, Malam (20%), Vizhi (15%), Sparisam (10%) were affected.
- Naadi - PithaVathanaadi (65%) was most commonly observed.
- In Neikuri examination, 50% Pithaneer were observed.
- The Toxicological study of Pungampoo Chooranam revealed no toxicity.

- The pharmacological study shows Anti Diabetic Activity in streptozotocin induced diabetic rats.
- Urine sugar Fasting and Postprandial became normal in 95% and 90% of patients respectively.
- Regarding Blood sugar level, fasting and post prandial blood sugar reduced in 95% and 87.5% of the cases respectively.
- HbA_1C level improved in 75% of cases which shows Good control in Madhumegam.
- The clinical trial shows that there is significant improvement in clinical manifestations of Madhumegam.
- The Biostatistical analysis of the clinical trial shows significant p-value <0.001and hence the treatment was effective and significant.

CONCLUSION

CONCLUSION

- Madhumegam is primarily due to derangement of Pitha kutram.
- The trial medicine Pungampoo Chooranam predominating with Thuvarppu suvai, it neutralises the deranged pitham by Ethirurai Maruthuvam.
- Pungampoo Chooranam reveals no toxicity in animal models and hence proved to be safe in human subjects.
- From preclinical Pharmacological studies, Pungampoo Chooranam has Anti-Diabetic activity.
- No adverse effect was reported during the clinical study.
- Pungampoo Chooranam significantly reduced blood sugar level and also reduced clinical features of Madhumegam.
- Pungampoo Chooranam is cost effective.

Hence I conclude that Pungampoo Chooranam will be a better drug that can be used in the treatment of Madhumegam.

ANNEXURES

Tha Tamil Nadu Dr.M.G.R.Medical University

69,Anna Salai,Guindy,Chennai - 600 032.

This Certificate is awarded to Dr/~~Mr/Mrs~~.....J. Nisha.....

for participating as ~~Resource Person~~ / Delegate in the Seventeenth (XVII) Workshop on

" RESEARCH METHODOLOGY & BIOSTATISTICS "

FOR AYUSH POST GRADUATES & RESEARCHERS

Organized by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University from 15th to 19th June 2015.

Dr.N.KABILAN, M.D.(Siddha)
READER,DEPT.OF SIDDHA

Prof. **Dr.P.ARUMUGAM,** M.D.,
REGISTRAR i/c

Prof. **Dr.D.SHANTHARAM,** M.D., D.Diab.,
VICE - CHANCELLOR

Government Siddha Medical College
Department of Medicinal Botany

Dr.S.Sankaranarayanan M.Sc., M.Phil., Ph.D.,
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Head of the Department

6, Anna Arch Rd,
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Tamil Nadu 600106.

AUTHENTICATION CERTIFICATE

Based upon the organoleptic/macroscopic/microscopic examination of fresh/market sample, it is certified that the specimen given by Dr. J. Nisha BSMS studying MD (S), Government Siddha Medical College, Arumbakkam, Chennai is identified below

Binomial name: ***Pongamia pinnata*** **(L.) Pierre**

Family: Fabaceae

Synonym(s): *Pongamia glabra* Vent

Regional names: Tamil:Pungamaram, English: Indian beech tree

References: Flora of Presidency, Gamble, J. S

Dr. S. Sankaranarayanan M.Sc., M.Phil., Ph.D.,

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GSMC/MB-03/2016

Date:01.06.2016

CERTIFICATE

This is to certify that the project entitled "**TOXICITY EVALUATION OF *PUNGAMPOO CHOORNAM* BY ACUTE TOXICITY -OECD 423 AND SUB-ACUTE REPEATED DOSE ORAL TOXICITY STUDY- OECD 407 IN RATS**" has been approved by the IAEC of Sathyabama University, Chennai.

IAEC Approval No.: **SU/CLATR/IAEC/IV/023/2016**
Animal Sanctioned: *Rattus norvegicus* / Wistar albino rats
Male: 9; Female: 15; Total: 24 (Twenty Four)
Date: 5.3.2016

DR.B.SHEELA RANI
Chair Person

DR.R.ILAVARASAN
CPCSEA Main Nominee

LABORATORY ANIMAL TECHNOLOGY AND RESEARCH (CLATR) ★ CENTRE FOR
SATHYABAMA UNIVERSITY
Chennai - 600 119

CERTIFICATE

This is to certify that the project entitled "**PHARMACOLOGICAL EVALUATION OF ANTIDIABETIC POTENTIAL OF PUNGAMPOO CHOORNAM ON STREPTOZOTOCIN INDUCED DIABETIC RATS**" has been approved by the Institutional Animal Ethics Committee of Sathyabama University, Chennai.

IAEC Approval No.: **SU/CLATR/IAEC/VII/051/2016**
Principal Investigator: Dr. J. Nisha
Animal Sanctioned: *Rattus norvegicus* / Wistar albino rats
Male: 24; Total: 24 (Twenty Four)
Date: 05.10.2016

B. Sheela Rani.

DR. B. SHEELA RANI
Chairperson

DR. R. ILAVARASAN
CPCSEA Nominee

TOXICITY STUDY

ACUTE TOXICITY STUDY- OECD GUIDELINE – 423.

IAEC NO: SU/CLATR/IAEC/IV/023/2016.

Acute toxicity study of the study drug *Pungampoo Chooranam* was carried out as per OECD guideline (Organization for Economic Co-operation and Development) Guideline-423.

Animal

Healthy adult Wistar albino rat weighing between 170-200 g were used for the study. The animals were housed in poly propylene cages and were kept in well ventilated with 100% fresh air by air handling unit (AHU). A 12 light / dark cycle were maintained .Room temperature was maintained between 22 $\pm$ 2^0 C and relative humidity 50–65%. They were provided with food (Sai feeds, Bangalore, India) and water *ad libitum*. All the animals were acclimatized to the laboratory for 7 days prior to the start of the study.

The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

Acute toxicity Study

Acute toxicity study will be carried out in accordance with OECD guideline 423.[70] The animals were fasted overnight with free access to water. The study was conducted with single oral dose administration of *Pungampoo Chooranam*.

Animal Grouping

One group consist of 6 female rats were used for this study . The dose utilized for evaluation of acute toxicity study is about 2000 mg/kg higher than that of the therapeutic dose.

Animal Grouping

GROUP I: Animals received Test drug 2000 mg/kg (p.o)

The animals were fasted overnight (12- 16 hrs) with free access to water. The study was conducted with single oral administration of study drug*Pungampoo Chooranam* 2000mg/kg (p.o). The animals were observed continuously for first 72 h and then 14 days for emerging signs of behavioral changes, body weight changes and for mortality.

Occurrence of toxicity in animals were observed continuously for the first 4 to 24 h and observed periodically for the next 14 days. Observation includes the change in skin, fur, eyes and mucus membrane. Appearance of C.N.S,C.V.S and A.N.S related toxicity such as tremors, convulsions, sedation, steric behavior, respiratory distress, cardiovascular collapse, response to sensory stimuli, salivation, diarrhea, lethargy, sleep, coma and mortality were observed with special attention.

Body weight was recorded periodically. At the end of the experiment all animals were subjected for gross necropsy and observed for pathological changes.

SUB-ACUTE TOXICITY STUDY - OECD Guideline - 407. [71]

Animals

Healthy adult Wistar albino rat weighing between 170-200 g were used for the study. The animals were housed in poly propylene cages and were kept in well ventilated with 100% fresh air by air handling unit (AHU). A 12 light / dark cycle were maintained .Room temperature was maintained between 22 $\pm$ 2^{o} C and relative humidity 50–65%. They were provided with food (Sai feeds, Bangalore, India) and water *ad libitum*. All the animals were acclimatized to the laboratory for 7 days prior to the start of the study.

The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

Animal Grouping

Animals were divided into three groups of 06 animals each consist of 3 male and 3 female rats.

GROUP I **:** Animals received saline 5 ml/kg b.w (p.o)

GROUP II **:** Animals received low dose of test drug 200 mg/kg (p.o)

GROUP III **:** Animals received high dose of test drug 400 mg/kg (p.o)

The animals were randomly divided into control group and drug treated groups for two different doses viz. low dose (200 mg/kg b.w) and high dose (400 mg/kg b.w).

The animals were administrated with the study drug once daily for 28 days. The animals in group I (control group) received normal saline 5 ml/kg b.w.

The animals in group II received low dose of *Pungampoo Chooranam* 200 mg/kg b.w (p.o) and group III received high dose of *Pungampoo Chooranam* 400 mg/kg b.w (p.o).

The rats were weighed periodically and observed for signs of toxicity pertains to C.N.S, C.V.S, A.N.S including behavioral changes, food - water intake and morphological changes. At the end of 28th day, the animals were fasted for overnight with free access to water. On 29th day the animals were sacrificed with excess anesthesia. Blood samples were collected from aorta and stored in EDTA (ethylenediamine –tetra actate) for Hematological analysis and for serum generation for biochemical analysis.

The vital organs including heart, brain, lungs, spleen, kidneys, liver, stomach, testes, and ovary were harvested and carefully examined for gross lesions. The organs were preserved in 10% formalin for histopathological assessment and interpretation.

Hematological analysis

Blood samples were analyzed using established procedures and automated Bayer Hematology analyzer. Parameters evaluated include Packed Cell Volume (PCV), Red Blood Cells (RBC) count, White blood cell count (WBC), Platelet Count, Hemoglobin (Hb), Mean cell Haemoglobin Concentration (MCHC), Mean Red Cell Volume (MCV), Mean Cell Hemoglobin (MCH), Mean platelet volume (MPV), Neutrophils, Eosinophil's, Basophils, Lymphocytes and Monocytes.

Biochemical analysis [72]

Serum samples were analyzed for High Density Lipoprotein (HDL), Low density Lipoprotein (LDL) , Very low density Lipoprotein (VLDL) , Triglycerides (TGL), Total Cholesterol , Blood urea nitrogen (BUN), Creatinine, Albumin, Total Protein, Glucose, Uric acid, Aspartate Transaminase (AST), Alanine amino Transaminase (ALT) and Alkaline Phosphatase (ALP) using Mind ray auto analyzer model BS 120.

Histopathological evaluation [73]

Organs included of heart, brain, lungs, spleen, kidneys, liver, stomach, testes and ovary. Histological slides of organs were made and observed under the microscope. The pathological observations of cross section of these organs were performed on gross and microscopic bases. Histological examinations were performed

on the preserved tissues with particular emphasis on those which showed gross pathological changes.

Statistical analysis

The statistical analysis was carried by one way ANOVA (GRAPH PAD PRISM 5 computer program). Results were expressed as mean ± standard error .A statistical comparison was carried out using the Dunnet's test for the control and treatment group.

Acute Toxicity Study

Analysis	**Group I**
Consistency	Soft
Shape	Pointed Head
Colour	Greenish brown
Mucous Shedding	Absence
Blood Cells	Absent
Signs of Infection	None Observed

Sub-Acute Toxicity Study

Analysis	**Group I**	**Group II**	**Group III**
Consistency	Soft	Soft	Soft
Shape	Oblong	Pointed Head	Pointed Head
Colour	Greenish brown	Greenish brown	Greenish brown
Mucous Shedding	Absence	Absence	Absence
Blood Cells	Absent	Absent	Absent
Signs of Infection	None Observed	None Observed	None Observed

RESULTS

Assessment of clinical signs in rats treated with *Pungampoo Chooranam*on Acute toxicity study

Parameter	Group I
Clinical Signs Parameters for the duration of 14 days	Test Drug 2000mg/ Kg
Number of animals observed	6 Female
Lacrimation	Absence
Salivation	Absence
Animal appearance	Normal
Tonic Movement	Absence
Clonic Movement	Absence
Laxative action	Very Mild
Touch Response	Normal
Response to Sound	Normal Response
Response to Light	Normal Response
Mobility	Normal Response
Respiratory Distress	Nil
Skin Color	Normal
Stereotype behavior	Absence
Piloerection	Absence
Limb Paralysis	Absence
Posture	Normal
Open field behavior	Normal
Gait Balancing	Normal
Freezing Behaviour	Absent
Signs of Stress and Anxiety	None Observed
Muscular coordination	Normal
Muscle grip	Normal
Sedation	Absence
Social Behavior	Normal
Urine Analysis	No Abnormality
Urine Colour	Yellowish
Urine pH	7
Urine -Glucose	Absence
Urine -Ketones	Absence
Urine- Bilirubin	Absence
Urine-Blood Cells	Negative
Urine - Pus cells	Negative
Mortality	Nil

Quantitative data on the body weight of rats treated with *Pungampoo Chooranam* in Acute toxicity study

Group I	Before Treatment Weight in Gms	After Treatment Weight in Gms
Mean	172.8	175.5
Std. Deviation	3.312	3.619
Std. Error	1.352	1.478

Values are mean ± S.D (n = 6 per group). Control and treatment group were compared statistically using one way ANOVA followed by Dunnett's test.

Assessment of clinical signs in rats treated with *Pungampoo Chooranam*on Sub-Acute toxicity study

Parameter	Group I	Group II	Group III
Clinical Signs Parameters for the duration of 28 days	Control	Test Drug 200mg/ Kg	Test Drug 400mg/ Kg
Number of animals observed	3 Male and 3 Female	3 Male and 3 Female	3 Male and 3 Female
Lacrimation	Absence	Absence	Absence
Salivation	Absence	Absence	Absence
Animal appearance	Normal	Normal	Normal
Tonic Movement	Absence	Absence	Absence
Clonic Movement	Absence	Absence	Absence
Laxative action	Absence	Absence	Very Mild
Touch Response	Normal	Normal	Normal
Response to Sound	Normal Response	Normal Response	Normal Response
Response to Light	Normal Response	Normal Response	Normal Response
Mobility	Normal	Normal	Normal
Respiratory Distress	Nil	Nil	Nil
Skin Color	Normal	Normal	Normal
Stereotype behavior	Absence	Absence	Absence
Piloerection	Absence	Absence	Absence
Limb Paralysis	Absence	Absence	Absence
Posture	Normal	Normal	Normal

Parameter	Group I	Group II	Group III
Open field behavior	Normal	Normal	Normal
Gait Balancing	Normal	Normal	Normal
Freezing Behaviour	Absent	Absent	Absent
Sings of Stress and Anxiety	None Observed	None Observed	None Observed
Muscular coordination	Normal	Normal	Normal
Muscle grip	Normal	Normal	Normal
Sedation	Absence	Absence	Absence
Social Behavior	Normal	Normal	Normal
Urine Analysis	No Abnormality	No Abnormality	No Abnormality
Urine Colour	Yellowish	Pale yellowish	Pale yellowish
Urine pH	6	7	7
Urine - Glucose	Absence	Absence	Absence
Urine - Ketones	Absence	Absence	Absence
Urine- Bilirubin	Absence	Absence	Absence
Urine-Blood Cells	Negative	Negative	Negative
Urine - Pus cells	Negative	Negative	Negative
Mortality	Nil	Nil	Nil

Effect of *Pungampoo Chooranam* on Body weight of Rats in Sub-acute toxicity study

Group I	**Before Treatment Weight in Gms**	**After Treatment Weight in Gms**
Mean	177.3	184.5
Std. Deviation	5.82	6.348
Std. Error	2.376	2.592
Group II	**Before Treatment Weight in Gms**	**After Treatment Weight in Gms**
Mean	186.5	198.7
Std. Deviation	5.32	5.715
Std. Error	2.172	2.333

Group III	Before Treatment	After Treatment Weight in Gms
Mean	183	196.3
Std. Deviation	5.514	6.088
Std. Error	2.251	2.486

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Quantitative data on the food and water intake of rats treated with *Pungampoo Chooranam* for 28 days in Sub-acute toxicity study

GROUP I	Food intake	Water intake
Mean	17.75	29.92
Std. Deviation	0.5693	0.9574
Std. Error	0.2846	0.4787
GROUP II	**Food intake**	**Water intake**
Mean	17.75	29.92
Std. Deviation	3.775	1.101
Std. Error	1.887	0.5507
GROUP III	**Food intake**	**Water intake**
Mean	20.08	34.42
Std. Deviation	3.957	2.267
Std. Error	1.978	1.133

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Pungampoo Chooranam* on Haematology profile of rats in sub-acute toxicity study

GROUP I	**WBC count ($\times 10^3$ µl)**	**RBC ($\times 10^6$ µl)**	**PLT ($\times 10^3$ µl)**	**MCV (fl)**	**MCH (pg)**	**MCHC (g/dl)**	**HGB (g/dl)**
Mean	12.23	5.817	918.7	60.92	19.82	31.42	11.07
Std. Deviation	2.719	0.9683	71.46	2.062	2.04	1.292	1.507
Std. Error	1.11	0.3953	29.17	0.842	0.8328	0.5275	0.6152
GROUP II	**WBC count ($\times 10^3$ µl)**	**RBC ($\times 10^6$ µl)**	**PLT ($\times 10^3$ µl)**	**MCV (fl)**	**MCH (pg)**	**MCHC (g/dl)**	**HGB (g/dl)**
Mean	11.12	7.783	710	58.97	20.22	32.48	11.17
Std. Deviation	1.046	1.055	279.5	3.893	3.268	1.393	1.508
Std. Error	0.4269	0.4308	114.1	1.589	1.334	0.5689	0.6157
GROUP III	**WBC count ($\times 10^3$ µl)**	**RBC ($\times 10^6$ µl)**	**PLT ($\times 10^3$ µl)**	**MCV (fl)**	**MCH (pg)**	**MCHC (g/dl)**	**HGB (g/dl)**
Mean	11.73	6.15	725.2	59.7	19.3	31.15	12.57
Std. Deviation	2.858	0.7064	299.3	3.3	2.467	1.924	1.94
Std. Error	1.167	0.2884	122.2	1.347	1.007	0.7856	0.7919

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Pungampoo Chooranam* on Haematology profile of rats in sub-acute toxicity study.

GROUP I	**Lymph (%)**	**Mon (%)**	**Neutrophils (X 10^3/mm^3)**	**Eosinophils (%)**	**Basophils (%)**	**MPV (fl)**
Mean	69.98	2.633	2.083	1.283	0.3333	6.483
Std. Deviation	3.602	1.102	0.9453	0.2639	0.5164	1.003
Std. Error	1.47	0.4499	0.3859	0.1078	0.2108	0.4094
GROUP II	**Lymph (%)**	**Mon (%)**	**Neutrophils (X 10^3/mm^3)**	**Eosinophils (%)**	**Basophils (%)**	**MPV (fl)**
Mean	78.53	2.15	2.233	1.333	0.5	6.1
Std. Deviation	7.821	0.9874	0.7394	0.2582	0.5477	1.468
Std. Error	3.193	0.4031	0.3018	0.1054	0.2236	0.5994
GROUP III	**Lymph (%)**	**Mon (%)**	**Neutrophils (X 10^3/mm^3)**	**Eosinophils (%)**	**Basophils (%)**	**MPV (fl)**
Mean	77.45	3.65	1.883	1.417	0.5	6.117
Std. Deviation	8.302	1.093	0.736	0.2041	0.5477	1.373
Std. Error	3.389	0.4463	0.3005	0.08333	0.2236	0.5606

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Pungampoo Chooranam* on Serum Bio-chemistry profile of rats in sub-acute toxicity study

GROUP I	**Blood sugar ® (mg/dl)**	**BUN (mg/dl)**	**Serum creatinine (mg/dl)**	**Serum total cholesterol (mg/dl)**	**Serum triglycerides level (mg/dl)**	**Serum HDL cholesterol (mg/dl)**	**Serum LDL cholesterol (mg/dl)**	**Serum VLDL cholesterol (mg/dl)**
Mean	82.33	19.67	0.7167	122.7	74.5	59.17	55	14.43
Std. Deviation	13.75	2.338	0.2927	6.088	10.41	15.88	7.849	3.189
Std. Error	5.613	0.9545	0.1195	2.486	4.249	6.483	3.204	1.302
GROUP II	**Blood sugar ® (mg/dl)**	**BUN (mg/dl)**	**Serum creatinine (mg/dl)**	**Serum total cholesterol (mg/dl)**	**Serum triglycerides level (mg/dl)**	**Serum HDL cholesterol (mg/dl)**	**Serum LDL cholesterol (mg/dl)**	**Serum VLDL cholesterol (mg/dl)**
Mean	82.5	13.33	0.5667	111	87	61	31	17.73
Std. Deviation	12.69	2.875	0.2251	16.6	10.26	13.81	17.66	3.333
Std. Error	5.182	1.174	0.09189	6.777	4.187	5.639	7.211	1.361
GROUP III	**Blood sugar ® (mg/dl)**	**BUN (mg/dl)**	**Serum creatinine (mg/dl)**	**Serum total cholesterol (mg/dl)**	**Serum triglycerides level (mg/dl)**	**Serum HDL cholesterol (mg/dl)**	**Serum LDL cholesterol (mg/dl)**	**Serum VLDL cholesterol (mg/dl)**
Mean	79.33	17.5	0.7667	115.3	66.83	71.33	41.5	14.85
Std. Deviation	11.59	2.074	0.2422	19.24	4.916	13.85	8.432	1.577
Std. Error	4.731	0.8466	0.09888	7.856	2.007	5.655	3.442	0.6438

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Effect of *Pungampoo Chooranam* on Serum Bio-chemistry profile of rats in sub-acute toxicity study

GROUP I	**Serum total protein (g/dl)**	**Serum albumin (g/dl)**	**(AST) (IU/ml)**	**(ALT) (IU/L)**	**(ALP) (IU/L)**
Mean	5.483	2.75	101.3	20.5	139.2
Std. Deviation	1.08	0.5648	20.53	2.881	58.25
Std. Error	0.4408	0.2306	8.381	1.176	23.78
GROUP II	**Serum total protein (g/dl)**	**Serum albumin (g/dl)**	**(AST) (IU/ml)**	**(ALT) (IU/L)**	**(ALP) (IU/L)**
Mean	4.967	3.783	88.17	39.33	163.3
Std. Deviation	1.172	0.7679	19.96	6.121	27.72
Std. Error	0.4787	0.3135	8.15	2.499	11.32
GROUP III	**Serum total protein (g/dl)**	**Serum albumin (g/dl)**	**(AST) (IU/ml)**	**(ALT) (IU/L)**	**(ALP) (IU/L)**
Mean	5.433	2.45	109.7	24.83	154
Std. Deviation	0.6713	0.6834	13.6	5.419	63.17
Std. Error	0.2741	0.279	5.554	2.212	25.79

Values are mean ± S.D (n = 6 per group of which 3 males and 3 females). Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

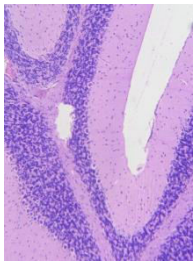
Quantitative data on absolute organ weight of rats treated with *Pungampoo Chooranam* for 28 days in Sub-acute toxicity study

GROUP I	**HEART (gms)**	**LIVER (gms)**	**KIDNEYS (gms)**	**SPLEEN (gms)**	**BRAIN (gms)**	**LUNG (gms)**	**STOMACH (gms)**	**TESTES (gms)**	**UTERUS & OVARY (gms)**
Mean	0.65	6.178	1.437	0.6	1.567	1.783	1.283	3.867	1.4
Std. Deviation	0.1467	0.5603	0.2587	0.1673	0.1862	0.3189	0.3869	0.4041	0.1
Std. Error	0.05989	0.2288	0.1056	0.06831	0.07601	0.1302	0.1579	0.2333	0.05774
GROUP II	**HEART (gms)**	**LIVER (gms)**	**KIDNEYS (gms)**	**SPLEEN (gms)**	**BRAIN (gms)**	**LUNG (gms)**	**STOMACH (gms)**	**TESTES (gms)**	**UTERUS & OVARY (gms)**
Mean	0.6283	6.022	1.487	0.5667	1.633	1.6	1.333	3.033	1.133
Std. Deviation	0.06242	1.324	0.3024	0.1366	0.1633	0.1414	0.3011	0.3786	0.05774
Std. Error	0.02548	0.5405	0.1235	0.05578	0.06667	0.05774	0.1229	0.2186	0.03333
GROUP III	**HEART (gms)**	**LIVER (gms)**	**KIDNEYS (gms)**	**SPLEEN (gms)**	**BRAIN (gms)**	**LUNG (gms)**	**STOMACH (gms)**	**TESTES (gms)**	**UTERUS & OVARY (gms)**
Mean	0.7967	4.902	1.458	0.7	1.633	1.833	1.5	3.033	1.4
Std. Deviation	0.07174	0.3578	0.3156	0.228	0.2338	0.2805	0.395	0.4163	0.1
Std. Error	0.02929	0.1461	0.1289	0.09309	0.09545	0.1145	0.1612	0.2404	0.05774

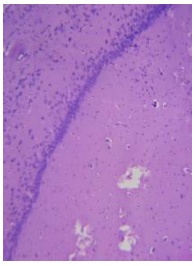
Values are mean ± S.D (n = 6 per group of which 3 males and 3 females) for Heart, Liver, Kidney, Brain, Spleen, Lung, Stomach. Values are mean ± S.D (n = 3 per group per sex) for testes , ovary and uterus for Control and treatment groups were compared statistically using one way ANOVA followed by Dunnett's test.

Histopathology of Brain (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I



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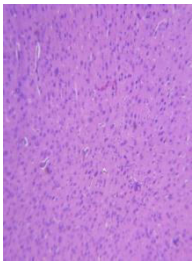
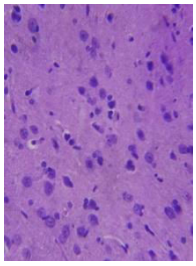
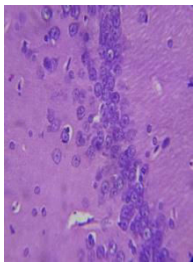


GROUP III

High Power Magnification 40X



GROUP I



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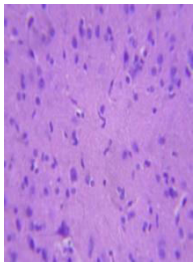
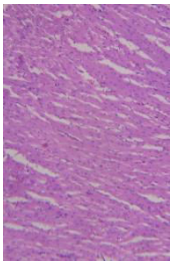


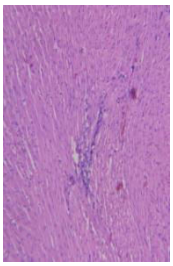
GROUP III

Histopathology of Heart (Female Rat) in Sub-acute toxicity Study

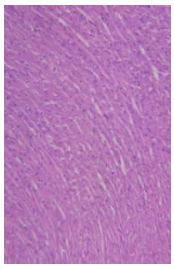
Low Power Magnification 10X



GROUP I



GROUP II



GROUP III

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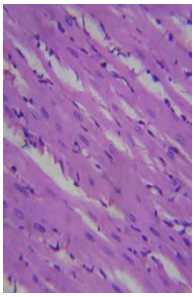
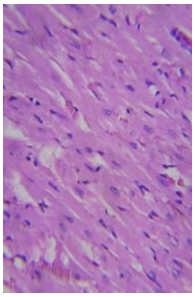
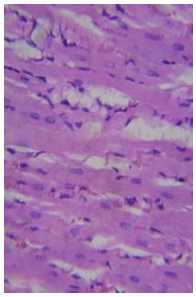
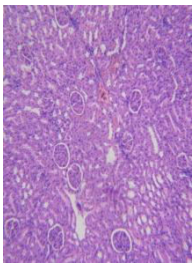
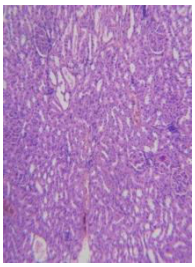
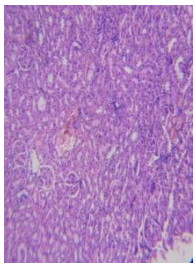




GROUP I **GROUP II** **GROUP III**

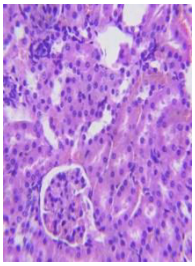
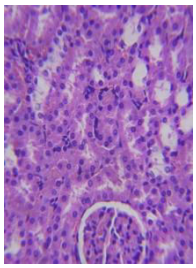
Histopathology of Kidney (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X

GROUP I **GROUP II** **GROUP III**

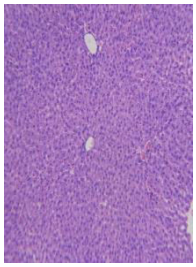
High Power Magnification 40X

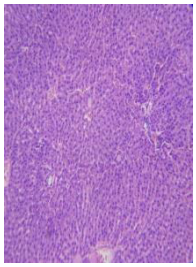

GROUP I **GROUP II** **GROUP III**

Histopathology of Liver (Female Rat) in Sub-acute toxicity Study

Low Power Magnification 10X



GROUP I



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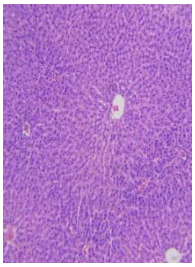
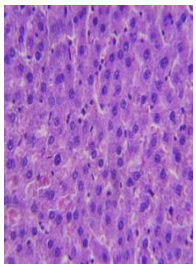
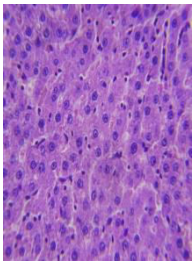


GROUP III

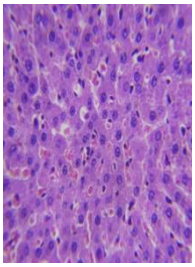
High Power Magnification 40X



GROUP I



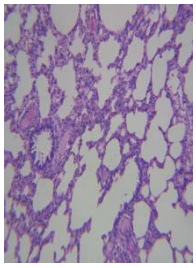
GROUP II



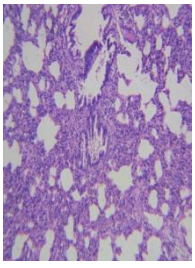
GROUP III

Histopathology of Lung (Female Rat) in Sub-acute toxicity Study

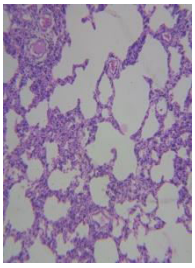
Low Power Magnification 10X



GROUP I

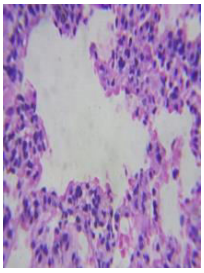


GROUP II

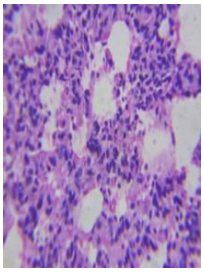


GROUP III

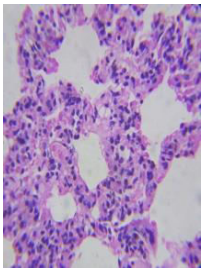
High Power Magnification 40X



GROUP I



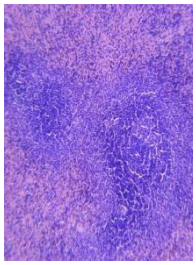
GROUP II



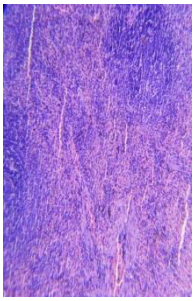
GROUP III

Histopathology of Spleen (Female Rat) in Sub-acute toxicity Study

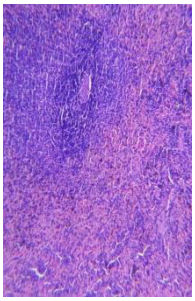
Low Power Magnification 10X



GROUP I

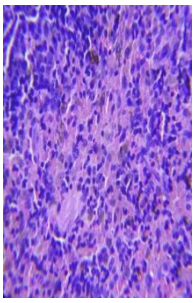


GROUP II

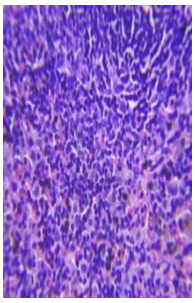


GROUP III

High Power Magnification 40X



GROUP I



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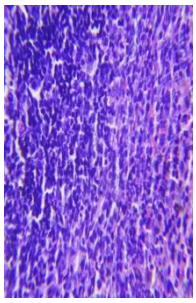
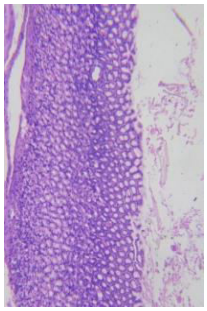


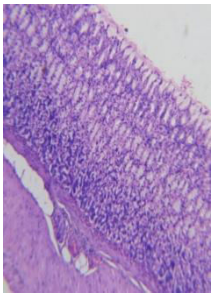
GROUP III

Histopathology of Stomach (Female Rat) in Sub-acute toxicity Study

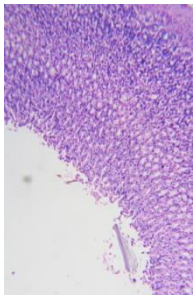
Low Power Magnification 10X



GROUP I

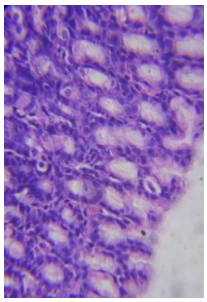


GROUP II

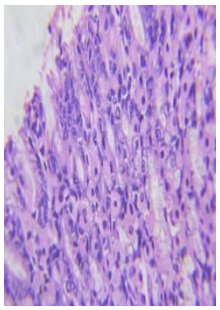


GROUP III

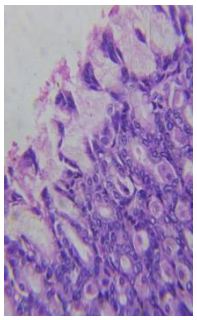
High Power Magnification 40X



GROUP I



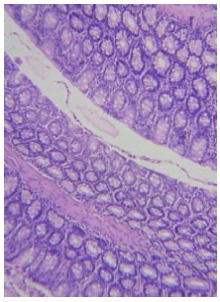
GROUP II



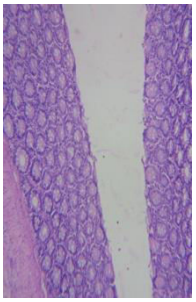
GROUP III

Histopathology of Uterus (Female Rat) in Sub-acute toxicity Study

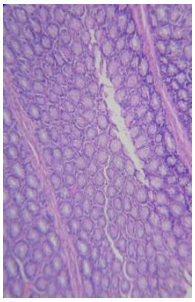
Low Power Magnification 10X



GROUP I

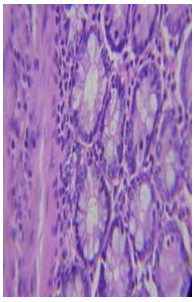


GROUP II

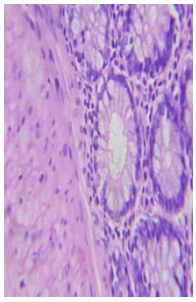


GROUP III

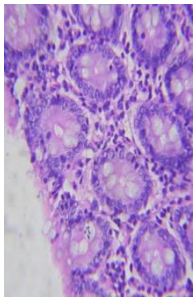
High Power Magnification 40X



GROUP I



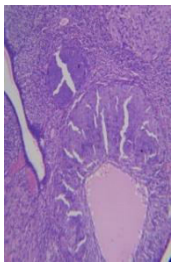
GROUP II



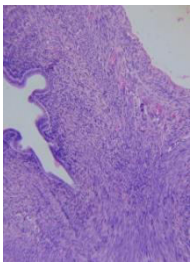
GROUP III

Histopathology of Ovary (Female Rat) in Sub-acute toxicity Study

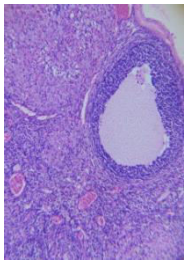
Low Power Magnification 10X



GROUP I

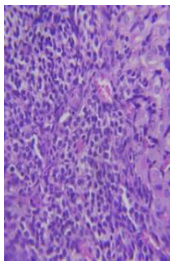


GROUP II

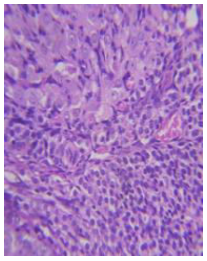


GROUP III

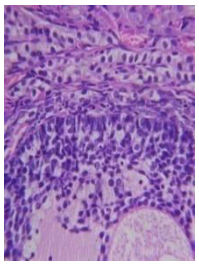
High Power Magnification 40X



GROUP I



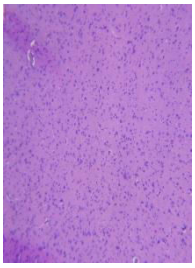
GROUP II



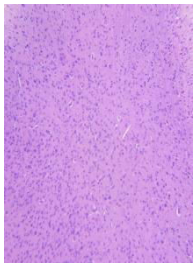
GROUP III

Histopathology of Brain (Male Rat) in Sub-acute toxicity Study

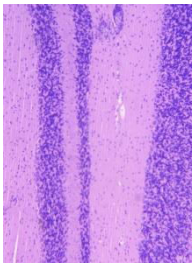
Low Power Magnification 10X



GROUP I

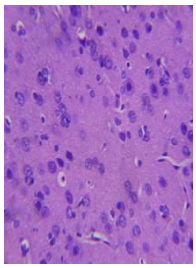


GROUP II

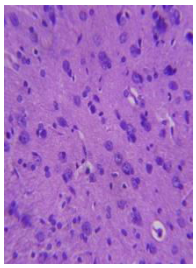


GROUP III

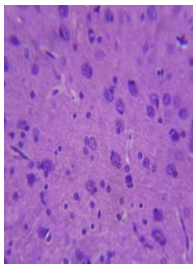
High Power Magnification 40X



GROUP I



GROUP II



GROUP III

Histopathology of Heart (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X

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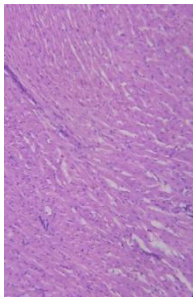
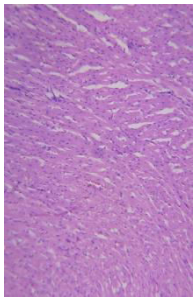
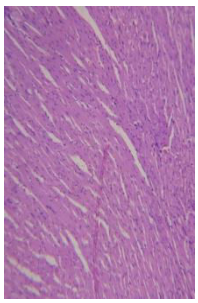


GROUP I

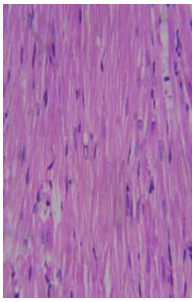


GROUP II

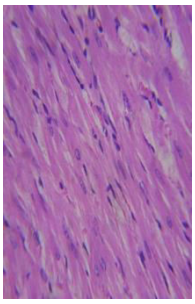


GROUP III

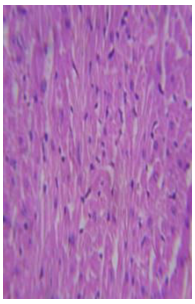
High Power Magnification 40X



GROUP I



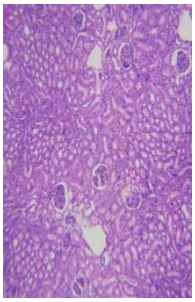
GROUP II



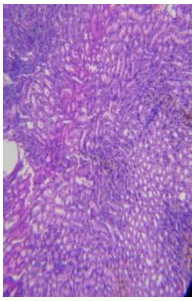
GROUP III

Histopathology of Kidney (Male Rat) in Sub-acute toxicity Study

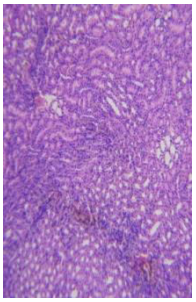
Low Power Magnification 10X



GROUP I

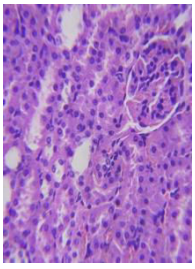


GROUP II

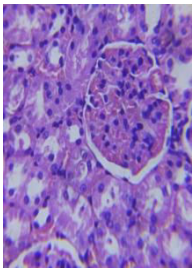


GROUP III

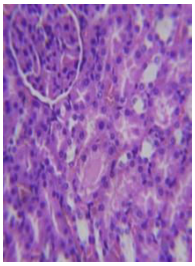
High Power Magnification 40X



GROUP I



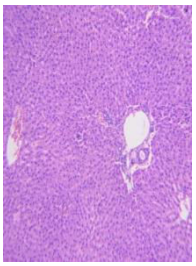
GROUP II



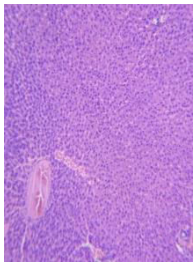
GROUP III

Histopathology of Liver (Male Rat) in Sub-acute toxicity Study

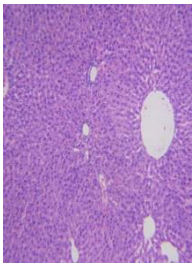
Low Power Magnification 10X



GROUP I

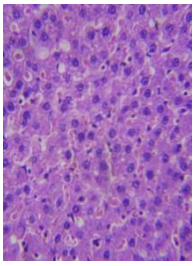


GROUP II

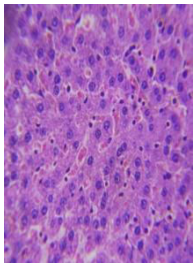


GROUP III

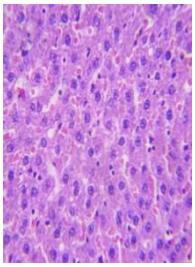
High Power Magnification 40X



GROUP I



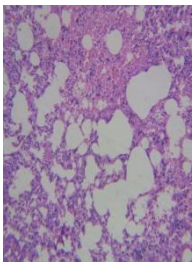
GROUP II



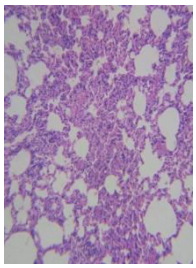
GROUP III

Histopathology of Lung (Male Rat) in Sub-acute toxicity Study

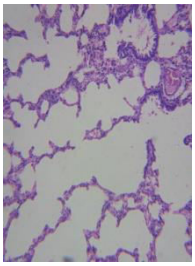
Low Power Magnification 10X



GROUP I

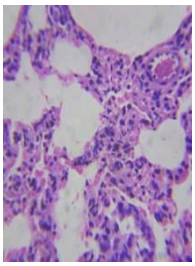


GROUP II

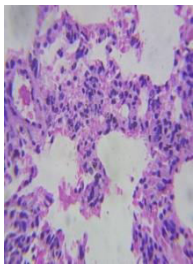


GROUP III

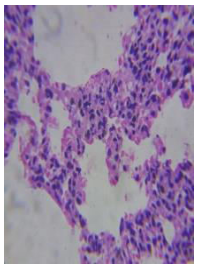
High Power Magnification 40X



GROUP I



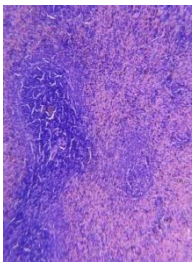
GROUP II



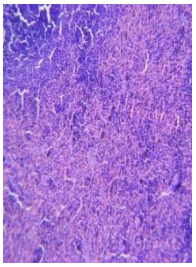
GROUP III

Histopathology of Spleens (Male Rat) in Sub-acute toxicity Study

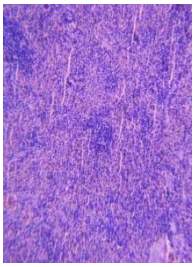
Low Power Magnification 10X



GROUP I

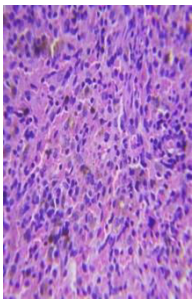


GROUP II

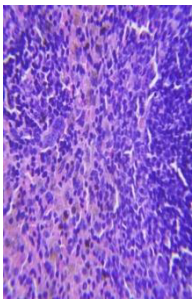


GROUP III

High Power Magnification 40X



GROUP I



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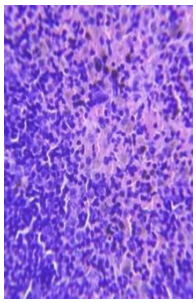
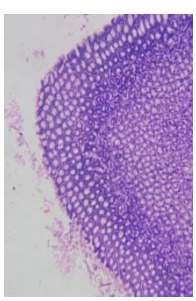
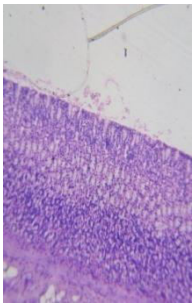
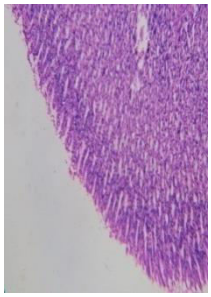


GROUP III

Histopathology of Stomach (Male Rat) in Sub-acute toxicity Study

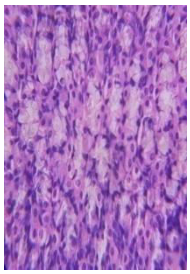
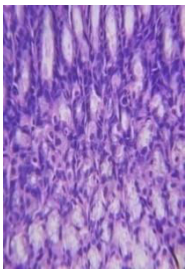
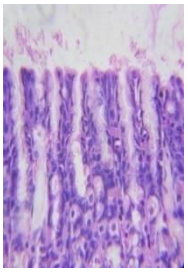
Low Power Magnification 10X

GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X

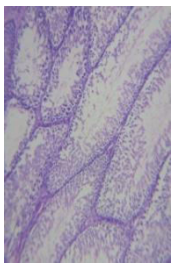
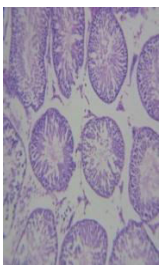
\

GROUP I **GROUP II** **GROUP III**

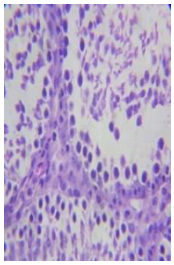
Histopathology of Testes (Male Rat) in Sub-acute toxicity Study

Low Power Magnification 10X

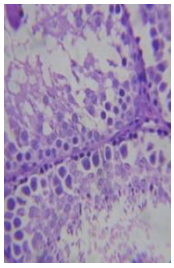




GROUP I **GROUP II** **GROUP III**

High Power Magnification 40X



GROUP I



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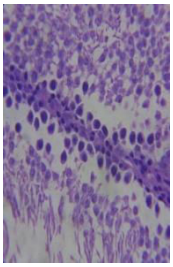


GROUP III

HISTOPATHOLOGY REPORT

BRAIN:

Neurons are very intact and there were no signs of edema or degeneration. No signs of ischemia or lesion were observed in sample belongs to group I,II and III.

LUNG:

Perivascular region appears normal, Alveolar septa and wall appeared widen and normal. No signs of airway secretion and bronchial secretion. Bronchial blood vessels and connective tissue appears normal with no signs of pulmonary edema were observed in both control and treated rats.

HEART:

Nucleus appears prominent with regular arrangement of fibres.No evidence on accumulation of adipose tissue on interstitium were observed in samples belongs to group I, II and III.

STOMACH:

Mucosal epithelium appears normal with no signs of ulceration.Lumina of blood vessels appears normal. Appearance of glandular lumen was normal in sample belongs to group I, II and III.

LIVER:

Hepatocyte appears with dark pigment chromatin in centri lobular and periportal region were observed in sample belongs to group I, II and III

SPLEEN:

Appearance of LF – lymphoid follicle; PALS – periarterial lymphoid sheath was normal with no significant signs of enlargement were observed in sample belongs to group I, II and III.

KIDNEY:

No evidence of lymphocytic infiltrate and inflammation. Epithelial lining on proximal convoluted tubule appears normal in sample belongs to group I,II and III.

TESTES:

Histo cytology of testicular tissue shows well differentiated germ cells with respect of spermatogonia includes spermatid and sperm were observed in sample belongs to group I,II and III.

UTERUS:

Appearance of endometrium, myometrium and uterine glands was normal. Arrangement of stratum basale, functionale and surface epithelium seems normal in samples belongs to group I, II and III.

OVARY:

Histopathological analysis of ovary showing normal corpus luteum (CL) and Primordial follicles with few mature ovarian follicles with no signs of abnormality. Appearance of antral follicle, primary oocyte and secondary follicles are normal in sample belong to group I, II and III.

PHARMACOLOGICAL STUDY

IAEC: SU/CLATR/IEAC/VII/051/2016

Animals

Healthy adult Wistar albino male rats weighing between 220-240 g were used for the study. The animals were housed in poly propylene cages and were kept in well ventilated with 100% fresh air by air handling unit. A 12 light / dark cycle were maintained .Room temperature was maintained between 22 $\pm$ 2^{o} Cand relative humidity 50–65%. They were provided with food (Sai feeds, Bangalore, India) and water *ad libitum*. All the animals were acclimatized to the laboratory for 7 days prior to the start of the study.The experimental protocol was approved by The Institutional Animal Ethics Committee of Sathyabama University, Chennai, Tamil Nadu, India.

Experimental Methodology

The animals were grouped into four groups of 6 animals each. Group I (Control group) -received normal saline,.Group II – Diabetic control rats administered with 45 mg/kg,i.p of STZ, Animals belongs to group III received 45 mg/kg,i.p of STZ and treated with 200mg/kg of *PungampooChooranam*. Animals belongs togroup IV received 45 mg/kg,i.p of STZ and treated with 400mg/kg of *PungampooChooranam*.

Induction of Diabetes

Streptozotocin (STZ), at a dose of 45 mg/kg body weight was dissolved in citrate buffer, injected intraperitoneally to induce diabetes. The animals will be fasted for 16hrs before prior to STZ injection, and after the injection 5% sucrose will be supplemented for 24hrs in order to prevent the animals from fatal hypoglycemia. One week after STZ injection, blood glucose level was checked using glucometer. The animals with a blood glucose level of more than 300 mg/dl were considered diabetic and included in the study.

Body Weight and Glucose estimation

The fasting blood glucose was measured on 0th, 14th and 28th day by glucose estimation strip. Body weight of the animals was measured before start of the study and also at the end of the study.

Sample Collection

At the end of the study, before sacrifice, the animals were fasted for overnight with free access to water. Animals were sacrificed with excess anesthesia. Blood samples were collected from retro orbital and cardiac puncture and stored in clot activator coated test tubes for serum biochemical analysis. Pancreas sample were harvested and carefully investigated for gross lesions.

Histopathology

A portion of pancreatic tissue was dissected out and fixed in 10% buffered neutral formal saline and processed. After fixation, tissues were embedded in paraffin. Fixed tissues were cut at 10 µm and stained with hematoxylin and eosin. The sections were examined under light microscope for histological changes.

Effect of *PungampooChooranam* on body weight of control and STZ induced experimental rats

	Before Treatment	**After Treatment**
GROUP I	**Body Weight in gms (0th day)**	**Body Weight in gms (28th Day)**
Mean	225.2	260.8
Std. Deviation	7.387	7.935
Std. Error	3.016	3.24
	Before Treatment	**After Treatment**
GROUP II	**Body Weight in gms (0th day)**	**Body Weight in gms(28th Day)**
Mean	221.5	188
Std. Deviation	7.287	4.94
Std. Error	2.975	2.017
	Before Treatment	**After Treatment**
GROUP III	**Body Weight in gms (0th day)**	**Body Weight in gms(28th Day)**
Mean	224.3	196.5
Std. Deviation	5.354	5.01
Std. Error	2.186	2.045
	Before Treatment	**After Treatment**
GROUP IV	**Body Weight in gms (0th day)**	**Body Weight in gms(28th Day)**
Mean	222.7	205.5
Std. Deviation	7.685	4.68
Std. Error	3.138	1.91

Effect of *PungampooChooranam* on oral glucose tolerance test of control and STZ induced experimental rats

	Blood glucose level (mg/dl)		
GROUP I	**0 Min**	**60 min**	**120 min**
Mean	77.67	142.2	124.3
Std. Deviation	6.532	5.382	3.615
Std. Error	2.667	2.197	1.476
	Blood glucose level (mg/dl)		
GROUP II treated with 200 mg/kg of	**0 Min**	**60 min**	**120 min**
Mean	79.33	135.5	118.8
Std. Deviation	5.502	5.089	2.714
Std. Error	2.246	2.078	1.108
	Blood glucose level (mg/dl)		
GROUP III	**0 Min**	**60 min**	**120 min**
Mean	79.17	126	105.8
Std. Deviation	4.355	4.427	5.231
Std. Error	1.778	1.807	2.136

Effect of *PungampooChooranam* on fasting blood glucose level and plasma insulin level of control and STZ induced experimental rats

Fasting Blood glucose level (mg/dl)				
GROUP I	**0th day**	**14th day**	**28th Day**	**Insulin (U/L)**
Mean	78.17	80.5	80.33	15.23
Std. Deviation	4.535	4.637	5.465	0.7607
Std. Error	1.851	1.893	2.231	0.3106
Fasting Blood glucose level (mg/dl)				
GROUP II	**0th day**	**14th day**	**28th Day**	**Insulin (U/L)**
Mean	75.5	283.7	311	5.917
Std. Deviation	6.473	20.48	17.7	0.4792
Std. Error	2.643	8.361	7.225	0.1956
Fasting Blood glucose level (mg/dl)				
GROUP III	**0th day**	**14th day**	**28th Day**	**Insulin (U/L)**
Mean	78.17	271.5	251.7	7.233
Std. Deviation	5.193	15.6	15.02	0.6186
Std. Error	2.12	6.371	6.13	0.2525
Fasting Blood glucose level (mg/dl)				
GROUP IV	**0th day**	**14th day**	**28th Day**	**Insulin (U/L)**
Mean	76.83	224.7	203.3	8.15
Std. Deviation	6.306	8.524	10.5	0.4593
Std. Error	2.574	3.48	4.287	0.1875

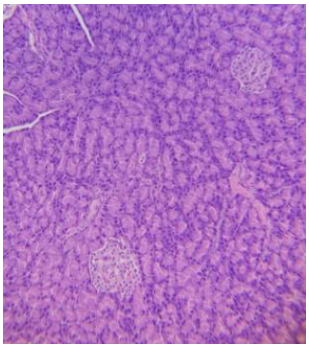
Effect of *PungampooChooranam*on HbA1C, serum urea and serum creatinine level of control and STZ induced experimental rats

GROUP I	**HbA1C (% Hb)**	**Serum Urea (mg/dl)**	**Serum Creatinine (mg/dl)**
Mean	6.583	24.67	0.5667
Std. Deviation	0.96	2.16	0.1862
Std. Error	0.3919	0.8819	0.07601
GROUP II	**HbA1C (% Hb)**	**Serum Urea (mg/dl)**	**Serum Creatinine (mg/dl)**
Mean	12.98	70.17	1.343
Std. Deviation	0.8954	4.622	0.2434
Std. Error	0.3655	1.887	0.09939
GROUP III	**HbA1C (% Hb)**	**Serum Urea (mg/dl)**	**Serum Creatinine (mg/dl)**
Mean	11.08	57.83	0.9433
Std. Deviation	0.9988	4.309	0.1461
Std. Error	0.4078	1.759	0.05965
GROUP IV	**HbA1C (% Hb)**	**Serum Urea (mg/dl)**	**Serum Creatinine (mg/dl)**
Mean	8.733	43.5	0.785
Std. Deviation	0.5125	5.128	0.1562
Std. Error	0.2092	2.094	0.06376

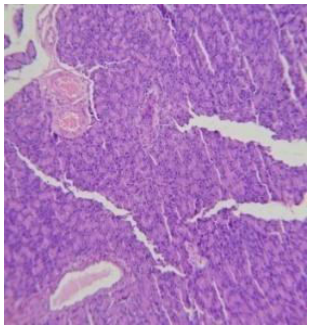
Histopathology of Rat Pancreas (H&E) Staining

Low Power Magnification 10 X

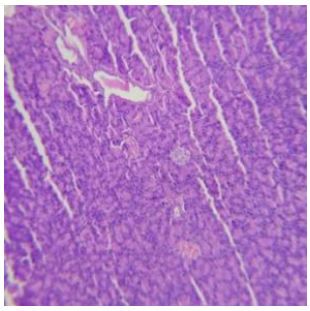
Control Group



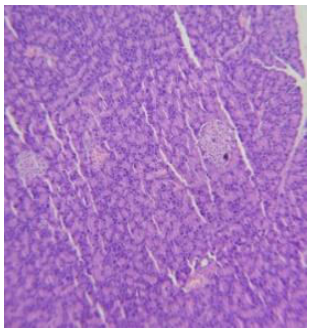
STZ Induced Group



STZ+ 200 mg/kg of *PungampooChooranam*



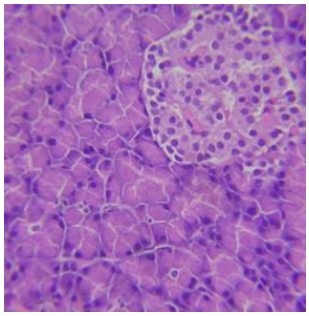
STZ+400 mg/kg of *PungampooChooranam*



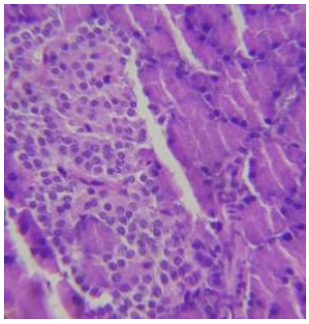
Histopathology of Rat Pancreas (H&E) Staining

High Power Magnification 40 X

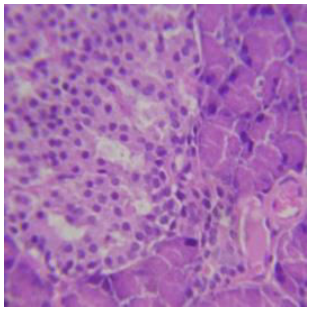
Control Group



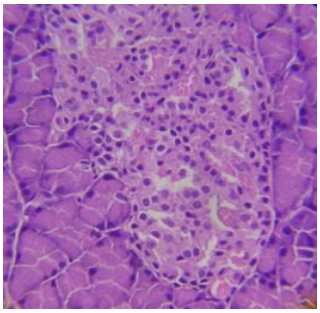
STZ Induced Group



STZ + 200 mg/kg of Pungampoo Chooranam



STZ + 400 mg/kg of Pungampoo Chooranam



PATHOLOGY REPORT

- Sample belongs to control group rat reveals normal histology of islet of Langerhans. Endocrine portion of acini zone appears normal with no signs of degeneration.
- Zone of fibrosis were observed with marginal loss of beta cells on islet of Langerhans were observed in sample belongs to group II rats. Deposition of collagen around inter lobular duct and vascular stroma was observed
- Apparent change in islets density was observed. Further there is a mild congestion of inter lobular blood vessels were observed occasional atrophic conditions of islet of Langerhans were observed.
- Almost normal density of beta cells were preserved in sample belongs to group IV rats with regular acini cellular zone and proper arrangement of islet of Langerhans were observed in sample belongs to group IV. Gradual restorations of pancreatic endocrine cells were observed in this group.

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Anna Govt. Hospital Campus, Arumbakkam, Chennai – 600106
Phone: 044-2621 4925, Fax: 044-2621 4809

20.1.2017

CERTIFICATE

Name of the student: Dr. J. Nisha, II year PG student, Pothu Maruthuvam, Government Siddha Medical College, Arumbakkam, Chennai-600 106.

Name of the sample: Pungampoo Chooranam

Name of the Experiment	I	II	Mean
Loss on drying(at 105°C)	9.22 %	9.26 %	9.24 %
Total ash	8.054 %	7.96 %	8.01 %
Water soluble ash	4.56 %	4.93 %	4.75 %
Acid insoluble ash	0.78 %	0.59 %	0.69 %
Water soluble extractive	26.48 %	26.43 %	26.46 %
Alcohol soluble extractive	29.17 %	29.52 %	29.35 %
n-Hexane soluble extractive	19.26%		
pH value (10%)	7.5		
TLC/HPTLC	Report Enclosed		

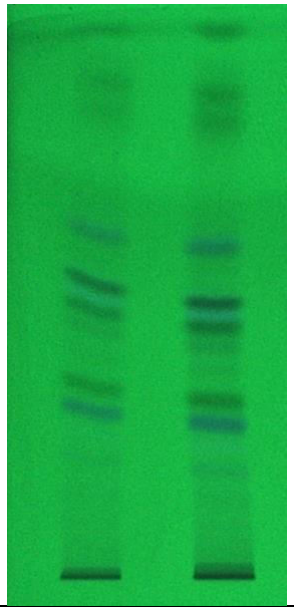
(R. Shakila)
Research Officer (Chemistry) & Head,
Department of Chemistry

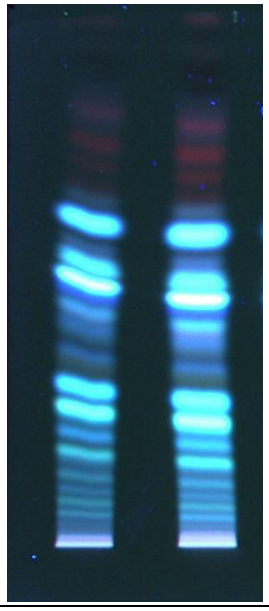
P. Elankani 20/1/17
(Dr. P. Elankani)
Research Officer (Scientist II) (Siddha)
for Assistant Director (Siddha) I/c

PUNGAMPOO CHOORANAM – CHLOROFORM EXTRACT

Stationary Phase - Silica Gel 60 F_{254}

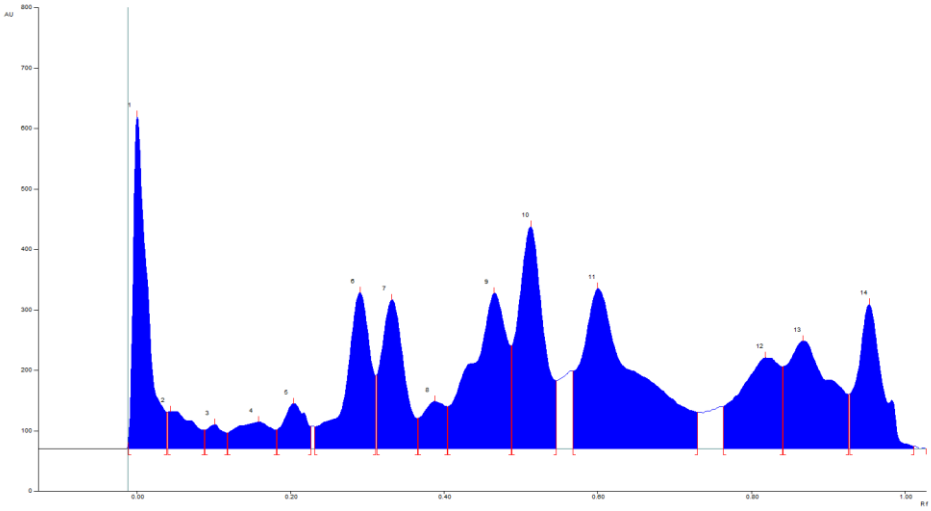
Mobile Phase - Toluene : Ethyl Acetate : Acetic Acid (5: 1.5: 0.25 v/v/v)





UV 254 nm		**UV 366 nm**		**whitelight 575 nm (Derivatized)**	
Color	**R_f value(s)**	**Color**	**R_f value(s)**	**Color**	**R_f value(s)**
Grey	0.12	Light Blue	0.06	Grey	0.06
Grey	0.18	Light Blue	0. 09	Pink	0.13
Dark blue	0.27	Blue	0. 11	Yellow	0. 27
Green	0.32	Light Blue	0.13	Blue	0.31
Grey	0.36	Bright Blue	0.18	Pink	0.34
Grey	0.42	Violet	0.21	Light blue	0.39
Green	0.48	Bright Blue	0.26	Light green	0. 44
Dark green	0.49	Bright Blue	0.27	Blue	0.48
Blue	0. 59	Violet	0.33	Pink	0.53
Grey	0.81	Violet	0.37	Yellowish green	0.58
Grey	0.87	Blue	0.41	Violet	0. 64
Grey	0.97	Bright Blue	0.46	Yellowish green	0. 79
		Bright Blue	0.50	Yellowish green	0.86
		Violet	0.53	Yellowish green	0.90
		Bright Blue	0.58		
		Blue	0.62		
		Dark Red	0.65		
		Dark Red	0.68		
		Bright Red	0.73		
		Pink	0.80		
		Violet	0.86		
		Violet	0.91		
		Pink	0.98		

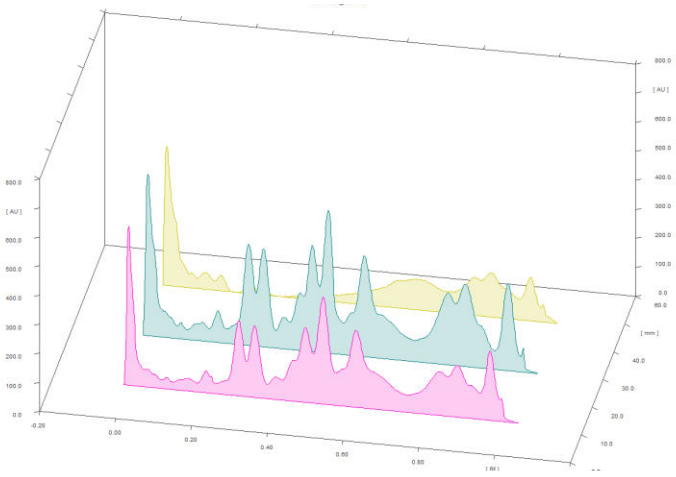
HPTLC Chromatogram @ 254 nm:



Peak Table @ 254 nm:

Peak	Start Position	Start Height	Max Position	Max Height	Max %	End Position	End Height	Area	Area %
1	-0.01 Rf	10.5 AU	0.00 Rf	549.4 AU	19.55 %	0.04 Rf	60.7 AU	8883.2 AU	9.54 %
2	0.04 Rf	60.8 AU	0.04 Rf	61.5 AU	2.19 %	0.09 Rf	30.9 AU	1849.8 AU	1.99 %
3	0.09 Rf	30.9 AU	0.10 Rf	39.8 AU	1.41 %	0.12 Rf	25.9 AU	764.3 AU	0.82 %
4	0.12 Rf	26.0 AU	0.16 Rf	44.5 AU	1.58 %	0.18 Rf	30.9 AU	1858.8 AU	2.00 %
5	0.18 Rf	31.2 AU	0.20 Rf	74.4 AU	2.65 %	0.23 Rf	37.3 AU	1916.9 AU	2.06 %
6	0.23 Rf	36.5 AU	0.29 Rf	258.5 AU	9.20 %	0.31 Rf	20.5 AU	7457.8 AU	8.01 %
7	0.31 Rf	122.0 AU	0.33 Rf	246.6 AU	8.77 %	0.37 Rf	50.1 AU	6497.1 AU	6.98 %
8	0.37 Rf	50.3 AU	0.39 Rf	78.4 AU	2.79 %	0.40 Rf	69.5 AU	2061.0 AU	2.21 %
9	0.40 Rf	69.5 AU	0.47 Rf	257.6 AU	9.17 %	0.49 Rf	70.2 AU	10589.2 AU	11.38 %
10	0.49 Rf	171.3 AU	0.51 Rf	367.7 AU	13.08 %	0.55 Rf	12.3 AU	10921.7 AU	11.74 %
11	0.57 Rf	128.8 AU	0.60 Rf	264.8 AU	9.42 %	0.73 Rf	59.9 AU	17233.8 AU	18.52 %
12	0.76 Rf	69.8 AU	0.82 Rf	150.3 AU	5.35 %	0.84 Rf	35.6 AU	7183.7 AU	7.72 %
13	0.84 Rf	135.9 AU	0.87 Rf	177.9 AU	6.33 %	0.93 Rf	89.9 AU	9027.8 AU	9.70 %
14	0.93 Rf	90.5 AU	0.95 Rf	238.7 AU	8.50 %	1.01 Rf	4.0 AU	6823.9 AU	7.33 %

3D Chromatogram @ 254 nm:



BIO-CHEMICAL ANALYSIS OF TRIAL MEDICINE

Preparation of Sodium Carbonate extract:

Trial drug (Pungampoo Chooranam) weighing 2 gms is mixed with 5 gms of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No	EXPERIMENT	OBSERVATION	INFERENCE
I	**TEST FOR ACID RADICALS**		
1(a)	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add 2ml of 4% Ammonium oxalate solution.	Absence of White Precipitate	Absence of Sulphate
1(b)	2ml of extract is added with 2ml of dilute hydrochloric acid until the effervescence ceases off. Then 2ml barium chloride solution is added.	Absence of White Precipitate	Absence of Sulphate
2	**Test for Chloride:** 2ml of extract is added with dilute nitric acid till the effervescence ceases. Then 2ml of silver nitrate solution is added.	Absence of white precipitate	Absence of Chloride
3	**Test for Phosphate** 2ml of the extract is treated with 2 ml of Ammonium molybdate solution and 2ml of concentrated nitric acid.	Absence of Yellow precipitate	Absence of Phosphate

4	**Test for Carbonate:** 2ml of the extract is treated with 2ml of magnesium sulphate solution.	Absence of white precipitate	Absence of Carbonate
5	**Test for Sulphide:** 1 gm of the substance is treated with 2ml of concentrated Hcl.	Absence of Rotten egg smelling	Absence of Sulphide
6	**Test for Nitrate:** 1gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube vertically down.	Absence of reddish brown gas.	Absence of Nitrate
7.a	**Test for Fluoride and Oxalate** 2ml of the extract is added with 2ml of dilute acetic acid and 2ml of calcium chloride solution and heated.	Absence of white precipitate	Absence of Fluoride and Oxalate
b.	5 drops of clear solution is added with 2ml of diluted sulphuric acid and slightly warmed to this, 1 ml of dilute potassium permanganate solution is added.	Absence of Discolourisation	Absence of Fluoride and Oxalate
8	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops of Acetic Acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absence of Nitrate
9	**Test for Borate** 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohol (95%) and introduced	Absence of Green tinged flame	Absence of Borate

II	**TEST FOR BASIC RADICALS**		
10	**Test for lead** 2 ml of the extract is added with 2 ml of Potassium iodide solution.	Absence of Yellow precipitate	Absence of Lead
11a	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non	Absence of Bluish green coloured flame.	Absence of Copper
b	2ml of the extract is added With excess of Ammonia solution	Absence of deep blue	Absence of Copper
12	**Test for Aluminium** To the 2 ml of extract. Sodium Hydroxide solution is added in drops to excess	Absence of White Precipitate.	Absence of Aluminium
13a	**Test for Iron** To the 2 ml of extract, 2 ml of Ammonium Thiocyanate Solution is added.	Absence of Blood red colour	Absence of Iron
b	To the 2 ml of extract, 2 ml Of Ammonium thiocyanate solution and 2 ml of concentrated Nitric	Absence of Blood red colour	Absence of Iron
14	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in	Presence of White Precipitate.	**Presence of Zinc**

15	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate	Absence of White Precipitate.	Absence of Calcium
16	**Test for Magnesium** 2ml of extract, Sodium	Absence of White Precipitate.	Absence of Magnesium
17	**Test for Ammonium** 2 ml of extract few ml of Nessler's Reagent and excess of Sodium	Absence of Reddish brown precipitate	Absence of Ammonium
18	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2ml of Cobalt Nitrate in 30% glacial	Presence of Yellow precipitate	**Presence of Pottasium**
19	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Absence of Yellow colour flame	Absence of Sodium
20	**Test for Mercury** 2 ml of the extract is treated with	Absence of yellow precipitate	Absence of Mercury

21	**Test for Arsenic** 2 ml of extract is treated with 2ml of silver Nitrate solution.	Absence of Yellow precipitate	Absence of Arsenic
22	**Test for Starch** 2ml of extract is treated with weak iodine solution	Absence of Blue colour	Absence of Starch
23	**Test of reducing Sugar** 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.	Presence of Green colour	**Presence of Reducing Sugar**
24	**Test of the alkaloids** 2ml of the extract is treated with 2ml of Potassium iodide solution.	Presence of Red colour	**Presence of Alkaloids**
25	**Test of the proteins** 2ml of the extract is treated with 2ml of 5% NaOH, mix well and add 2 drops of copper sulphate solution.	Absence of Violet colour	Absence of Proteins

RESULTS:

The given sample (Pungampoo Chooranam) contains,

Reducing Sugar, Alkaloids, Zinc and Potassium.

GOVERNMENT SIDDHA MEDICAL COLLEGE
Arumbakkam, Chennai-106

Communication Of The Decision Of Institutional Ethics Committee (IEC)

IEC No: GSMC-CH-ME-4/2015/009

Protocol title: A CLINICALSTUDYON MADHUMEGAM (DIABETES MELLITUS)WITH THE EVALUATION OF SIDDHA DRUG PUNGAMPOO CHOORANAM	
Principal Investigator:	DR.J. NISHA
Name & Address of Institution : Government siddha medical college, Arumbakkam, Chennai-106	
☑ New Review	☐ Revised Review ☐ Expedited Review
Date of review (DD/MM/YY):	26-03-2015
Date Of Previous Review, if Revised Application :	
Decision of the IEC	
☑ Recommended	☐ Recommended with suggestions
☐ Revision	☐ Rejected
Suggestions / Reasons / Remarks : 1. change duration from 90 days to 48 days. 2. HbA1C level should be 6.5 to 8%.	
Recommended for a period of 1 year from date of completion of preclinical studies:	

Please Note:

- Inform IEC immediately in case of any adverse events/serious drug reaction.
- Seek IECapproval in case of any change in the study procedure, site and investigator
- This approval is valid only for period mentioned above
- IEC member have the right to review the trial with prior intimation.

26/3/15.

Dr.P.Jeyaprakashnarayanan
Chairman

Dr.V. Banumathi
Member Secretary

INSTITUTIONAL ETHICS COMMITTEE

Date:

Sub: IEC review of research proposals.

Ref:Your letter dated

MEMBERS	PARTICIPATION	SIGNATURE
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DR.V.BANUMATHI M.D(S)., Member Secretary		
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DR.G.AADINAAATH REDDY,M.Pharm, Ph.D., Pharmacologist	✓	26/03/15
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DR.P.VIDYA M.B.B.S., DMRD., Modern Medicine Expert	✓	26/3/15
MR.P.SARAVANAN., Puplic Person	✓	

26/3/15.

Dr.P.Jeyaprakashnarayanan
Chairman

Dr.V.Banumathi
Member Secretary

CLINICAL PROGNOSIS

Treatment for Madhumegam:

The most popular non parametric statistical tool, namely, McNemar Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

S. No	Signs&Symptoms	Before Treatment	After Treatment
		n%	n%
1.	Polyuria	40(100)	7(17.5)**
2.	Polyphagia	40(100)	5(12.5)**
3.	Polydipsia	40(100)	4(10)**
4.	Itching present all over the body	4(10)	0(0)*
5.	Pain all over the body	34(85)	7(17.5)**
6.	Dryness of Mouth & Throat	40(10)	0(0)*
7.	Constipation	8(20)	0(0)*
8.	Emaciation	4(10)	3(7.5)
9.	Skin infection	4(10)	0(0)*
10.	Disturbed Sleep	40(100)	4(10)**

McNemat test, C.I: 95%, *P<0.05; **P<0.001

Software: spss17 version

Number of cases: 40

Inference:

Since the p value is significant in signs and symptoms except emaciation. So there is significant reducing of signs & symptoms except emaciation among the patients for the treatment of Madhumegam. Hence it is concluded that the treatment was effective and **significant.**

Effect of Pungampoo chooranam on Fasting Blood Sugar level in Madhumegam cases

S. no	Fasting blood sugar level in mg	
	Before Treatment	After Treatment
1.	132	110
2.	138	120
3.	136	122
4.	131	119
5.	136	94
6.	139	124
7.	132	121
8.	129	97
9.	133	109
10.	130	106
11.	127	103
12.	140	132
13.	129	98
14.	126	98
15.	139	105
16.	133	111
17.	137	113
18.	127	95
19.	129	119
20.	138	114
21.	133	120
22.	131	109
23.	128	94
24.	135	108
25.	138	122
26.	140	130
27.	136	121
28.	131	105
29.	128	108
30.	136	114
31.	128	106
32.	133	117
33.	130	102
34.	128	97
35.	138	119
36.	131	98
37.	136	112
38.	133	123
39.	131	113
40.	134	119

Software: spss17 version

Variables: Fasting Blood Sugar Level (mg) – before treatment, after treatment

Number of cases: 40

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.662

Before and after treatment mean difference ± SEM: 21.80 ± 1.28

P Value (2 tailed): $p<0.001$.

Inference:

Since the p value is significant ($p<0.001$). The hypothesis is not accepted. So there is significant reducing of Fasting blood sugar level (mg) among the patients for the treatment of Madhumegam. Hence it is concluded that the treatment was effective and significant.

Effect of Pungampoo chooranam on Postprandial blood Sugar level in Madhumegam cases.

S. no	Postprandial blood sugar level in mg	
	Before Treatment	After Treatment
1.	231	193
2.	233	191
3.	221	188
4.	202	168
5.	189	152
6.	242	218
7.	214	180
8.	230	188
9.	214	181
10.	219	186
11.	183	161
12.	263	217
13.	220	159
14.	227	161
15.	247	196
16.	188	143
17.	213	177
18.	185	157
19.	218	169
20.	220	173
21.	203	171
22.	226	180
23.	214	179
24.	245	208
25.	253	188
26.	258	203
27.	206	174
28.	186	166
29.	219	179
30.	226	187
31.	221	176
32.	228	179
33.	219	186
34.	228	176
35.	257	221
36.	184	168
37.	231	173
38.	222	182
39.	229	197
40.	237	181

Software: spss17 version

Variables: Postprandial Blood Sugar Level (mg) – before treatment, after treatment

Number of cases: 40

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.823

Before and after treatment mean difference ± SEM: 40.47 ± 1.85

P Value (2 tailed): $p<0.001$.

Inference:

Since the p value is significant ($p<0.001$). The hypothesis is not accepted. So there is significant reducing of postprandial blood sugar level (mg) among the patients for the treatment of Madhumegam. Hence it is concluded that the treatment was effective and significant.

Effect of Pungampoo chooranam on HbA1C level in Madhumegam cases.

S. no	HbA1C	
	Before Treatment	After Treatment
1.	7.1	6.4
2.	7.3	6.2
3.	7.0	6.3
4.	6.9	6.4
5.	6.6	5.2
6.	8.0	7.9
7.	7.3	6.5
8.	7.2	6.4
9.	7.3	6.3
10.	6.9	6.1
11.	6.7	5.8
12.	8.0	7.8
13.	7.8	6.5
14.	7.1	6.4
15.	7.8	6.3
16.	6.7	5.7
17.	7.0	6.5
18.	6.8	5.7
19.	7.3	6.5
20.	7.4	6.8
21.	6.8	6.4
22.	7.0	6.7
23.	8.0	7.9
24.	7.8	7.5
25.	7.2	7.0
26.	8.0	7.6
27.	6.8	6.3
28.	6.7	5.8
29.	7.3	6.5
30.	7.4	6.8
31.	7.6	7.3
32.	7.5	7.1
33.	6.8	6.1
34.	7.9	7.6
35.	8.0	7.8
36.	7.1	5.9
37.	7.6	6.9
38.	8.0	7.3
39.	7.0	6.4
40.	6.9	6.6

Software: spss17 version

Variables: HbA1C Level – before treatment, after treatment

Number of cases: 40

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.864

Before and after treatment mean difference ± SEM: 0.66 ± 0.06

P Value (2 tailed): $p<0.001$.

Inference:

Since the p value is significant ($p<0.001$). The hypothesis is not accepted. So there is significant reducing of HbA1C level among the patients for the treatment of Madhumegam. Hence it is concluded that the treatment was effective and significant.

CONSENT FORM

I certify that I have disclosed all the details about the study in the terms readily understood by the patient.

DATE: **SIGNATURE**

NAME

CONSENT BY THE PATIENT

I have been informed to my satisfaction by the attending physician the purpose of the clinical trial and the nature of the drug treatment and follow up including the lab investigations to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of the trial without having to give reasons for doing so.

I am exercising my free power of choice, and hereby give my consent to be included as a subject in the clinical trial of **PUNGAMPOO CHOORANAM** for the treatment of **MADHUMEGAM.**

DATE: **SIGNATURE**

NAME

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CASE SHEET PROFORMA

GOVERNMENT SIDDHA MEDICAL COLLEGE

POST GRADUATE DEPARTMENT – MARUTHUVAM BRANCH

CHENNAI – 600 106.

CASE SHEET PROFORMA FOR MADHUMEGAM

(NON INSULIN DEPENDENT DIABETES MELLITUS – NIDDM)

OP No / IP No	:		Nationality	: Indian
Ward No	:		Religion	:
Bed No	:		D.O.A	:
Name (In Block Letters)	:		D.O.D	:
Age	:		No of Days Treated	:
Sex	:	Male/ Female		
Occupation	:		Diagnosis	:
Income/Month	:		Result	:
Permanent Address	:			
Temporary Address	:	Govt. Siddha Medical College, Chennai – 600 106.		

1. Complaint and duration :

2. History of present illness :

3. History of previous illness :

4. Personal history

   Marital History :

   Occupation :

   Environment :

Social History :

Habits :

5. Family history :

SIDDHA ASPECT

GENERAL CONDITION ON ADMISSION

1. NILAM: 5

1. Kurinji
2. Mullai
3. Marutham
4. Neithal
5. Paalai

2. PARUVA KAALAM: 6

1. Kaar Kaalam : (Aavani, Purattasi)
2. Koothir Kaalam : (Ayppasi, Karthigai)
3. Munpani Kaalam : (Maarkazhi, Thai)
4. Pinpani Kaalam : (Maasi, Panguni)
5. Elavenil Kaalam : (Chittirai, Vaikasi)
6. Mudhuvenil Kaalam : (Aani, Aadi)

3. UDAL: 4

1. Vali Udal
2. Azhal Udal
3. Iya Udal
4. Kalappu Udal

4. KANMENTHIRIYANGAL: 5

1. Vaai
2. Kaal
3. Kai
4. Eruvai
5. Karuvai

5. PORI / PULANGAL: 5

1. Mei - Ooru
2. Vaai - Suvai
3. Kann - Oli
4. Mookku - Nattram
5. Sevi – Osai

6. GUNAM: 3

1. Sathuva Gunam
2. Rajo Gunam
3. Thamo Gunam

7. UDAL KATTUGAL: 7

1. Saaram
2. Senneer
3. Oon
4. Kozhuppu
5. Enbu
6. Moolai
7. Sukkilam / Suronitham

8. MALAM: 3

1. Malam
2. Moothiram
3. Viyarvai

9. MUKKUTRANGAL

VALI

1. Piraanan
2. Abaanan
3. Uthaanan
4. Viyaanan
5. Samaanan
6. Naagan
7. Koorman
8. Kirukaran
9. Devathathan
10. Dhananjeyan

AZHAL

1. Anala Pitham
2. Ranjaga Pitham
3. Aalosaga Pitham
4. Praasaga Pitham
5. Saathaga Pitham

IYAM

1. Avalambakam
2. Kilethagam
3. Pothagam
4. Tharpagam
5. Santhigam

10. ENVAGAI THERVU

1.Naadi

2.Sparisam

3.Naa

4.Niram

5.Mozhi

6.Vizhi

7.Malam:

I. Niram
II. Irugal
III. Ilagal

8. Moothiram:

I. Neerkuri:
    i. Niram
    ii. Edai
    iii. Manam
    iv. Nurai
    v. Enjal
II. Neikuri:

MODERN METHODS:

General Examination:

- Consciousness and Intelligence
- Voice and Speech
- General appearance
- Height and Weight
- Anaemia
- Cyanosis
- JVP
- Jaundice
- Clubbing
- Ascites
- Oedema
- Lymphadenopathy
- Temperature
- Respiration
- Pulse
- Blood Pressure

INVESTIGATION

A) BLOOD INVESTIGATIONS:

BLOOD INVESTIGATIONS		**BEFORE TREATMENT**	**AFTER TREATMENT**
Blood glucose (mg/dl)	F		
	PP		
Renal Function Test	Blood Urea		
	Serum creatinine		
Lipid Profile	Serum Cholestrol		

B) URINE INVESTIGATIONS:

URINE INVESTIGATIONS	**BEFORE TREATMENT**	**AFTER TREATMENT**
Urine Sugar (F)		
Urine Sugar (PP)		

C) SPECIFIC INVESTIGATIONS:

HbA1C	**BEFORE TREATMENT**	**AFTER TREATMENT**

D) **BODYMASS INDEX (BMI)**

SIGNS AND SYMPTOMS:

S.No	CLINICAL FEATURES	BEFORE TREATMENT	DURATION TREATMENT						AFTER TREATMENT
			14th Day	21st Day	28th Day	35th Day	42nd Day	49th Day	
1	Polyuria								
2	Polyphagia								
3	Polydipsia								
4	Itching All Over The Body								
5	Dryness Of The Mouth And Throat								
6	Constipation								
7	Disturbed Sleep								
8	Pain All Over The Body								
9	Skin Infection								
10	Emaciation								
11	Glycosuria								

Others specify, if any:

DIAGNOSIS

MADHUMEGAM (TYPE II DIABETES MELLITUS)

Trial Drug : PUNGAMPOO CHOORANAM

Dose : 2 Gram

Anubanam : WARM WATER

Duration of Treatment : 48 days

DATE	WEEKLY REPORT	MEDICINE

ADVICE:

Medical Officer: **H.O.D/Guide**

DISCHARGE SHEET

GOVERNMENT SIDDHA MEDICAL COLLEGE,
POST GRADUATE DEPARTMENT
MARUTHUVAM BRANCH, CHENNAI – 600 106.

Proforma for Madhumegam

IP NO	:	Date of Admission	:
Name	:	Date of Discharge	:
Age / Sex	:	No of days Treatment	:
Occupation	:	Diagnosis	:
Income:		Result	:
Nationality	:		
Religion	:		

Patient condition on admission	On discharge
Complaints and duration	
Pulse	
Weight	
Blood Pressure	
Blood Sugar	
Urea	
Serum Cholesterol	
Urine Sugar	

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